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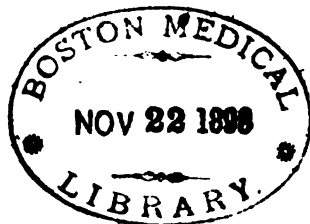
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A CLINICAL LECTURE

ON

DIVERS' PARALYSIS AND LEAD PARALYSIS.

Delivered at Guy's Hospital, February 5th, 1898,

BY

FREDERICK TAYLOR, M.D., F.R.C.P., M.R.C.S.,
Physician and Lecturer on Medicine at the Hospital.

GENTLEMEN,—The case I wish to mention to you to-day is of exceptional interest. It is one of a complaint known as divers' paralysis, or caisson disease. It has long been known that professional divers, that is, men who earn their living by diving to considerable depths in the sea in order to recover valuables which have been sunk, and to attend to defective piers and buttresses of bridges, are liable to suffer from certain symptoms under given conditions immediately in connection with their occupation, and obviously related in some way to the change of pressure to which they are subjected in their transference from the lower levels of the water to the upper air. This patient was admitted into the hospital on January 22nd for weakness in his legs. He is a man aged forty-three, and he was a very fine specimen of a man, weighing once 19 st., and now 17 st. 7 lbs. He was born in Canada, but while quite young he came to England and became a sailor, following that occupation five years, during which time he sailed to nearly all parts of the world. For the past seventeen years he has been a diver in the service of private firms. He told us that divers who are in the employ of private firms have to go to greater depths than those under government. Ten years ago he had scurvy. He has had gonorrhœa three or four times, and denies syphilis. He has already had three attacks of paralysis similar to that from which he is now suffering, the first being in 1890, when he was in the hospital under Dr. Pye-Smith for three months. That attack was very much

like the present one, consisting of weakness of the legs and slight impairment of sensation. He recovered completely. The second attack was in 1893, and it came on after diving at a depth of 162 feet off the coast of Florida. The patient felt sick, and when pulled up into the lighter he lost the power of his legs, so that he could not walk. When in the hospital he suffered from insomnia, and he several times had a drachm of laudanum to induce sleep. His reflexes and sensation were this time normal, and the electrical reactions were also normal. He went out in three weeks' time, against our advice. However, he soon got strong again, and was at work again shortly after leaving the hospital.

His next attack occurred two years later, after diving 175 feet off Sandy Hook, which is the greatest depth at which he has worked. The divers on that occasion were down about five minutes, and five minutes were occupied in the ascent to the surface. He lost consciousness, and on getting into the lighter was almost completely paralysed. He soon recovered consciousness, but the other man with whom he was working died without recovering consciousness. During the ascent the patient had flashes of light across the eyes, and noises in his ears.

Besides these attacks the patient has had very bad attacks of cramp, and sometimes great abdominal pain. They soon pass off, but he has to quit work while they last. After diving, he sometimes has attacks of wrist-drop and foot-drop. Otherwise he has enjoyed good health.

The history of the present illness is as follows:—He was working with five other men at a depth of 162 feet, and went down three times, remaining below twenty minutes each time; the ascent occupied six minutes. He felt quite well after these descents, but at the fourth descent a heavy piece of machinery slipped and jammed him up against an iron beam. He did not lose consciousness, but tried to economise the air at his disposal. After about five minutes he felt suffocated, had intense pain and noises in the head, and began to lose consciousness. It had been noticed on the lighter that the air-tube was not working, and another diver went down, and by following the line reached our patient and freed the air-tube, so that he could be drawn up. It is interesting to note that on this occasion the ascent occupied only one minute.

He felt very giddy and sick, but did not notice anything else amiss. On standing he noticed a numb feeling in his feet, and this was present also in his arms, both as to power and sensation. This all happened nine weeks before admission here, during which time he has been treated in America, mostly by quacks. The patient regards this attack as his worst, and has come over to England specially to be treated at Guy's Hospital. He has no pain, but he feels giddy. There has never been any incontinence of urine or incontinence of fæces, but sometimes he has to pass water very frequently. He is unable to walk up or down stairs without assistance. The numbness in the feet has now passed away, but he complains of a pricking sensation in them. Sometimes he is communicative and cheerful, and at other times the reverse. There is marked loss of power in both his legs, and he can be easily prevented from flexing his knees, while the power of extension is also diminished. There is no loss of power elsewhere. There is no marked wasting of the muscles of the legs, though they are soft and flabby; the patient does not think his legs are smaller. The plantar reflexes are present, and the soles of the feet are especially sensitive. The knee-jerks are present and normal, and there is no ankle-clonus. There is partial anæsthesia over the inner side of both legs, from three inches above the knees to three inches above the ankles, the areas being roughly symmetrical; and in them the patient frequently does not feel a touch. There is no loss of sensation to pain. Heat and cold sensations are normal, and the sensation in other parts of the body is normal. The respiratory, circulatory, and urinary systems are normal.

In spite of his having come all the way to Guy's Hospital to be treated, he left again after a very short stay and before he had completely recovered.

The case offers many points of interest. The disease is one which is well known and recognised, but it still presents some problems in regard to its pathology. The patient gave us some interesting information as to the difficulties to which people engaged in this occupation are liable to. The man says the paralysis only comes on after working at great depths, and seems to be due to too sudden alterations of pressure. The disease is frequently due to divers having to be quickly hauled up,

owing to the air-tube being foul. You will remember that this patient was hauled up in one minute from a depth of 160 feet on one occasion, the same ascent usually occupying five or six minutes. The patient says old men are more liable to these attacks than younger ones, and that the character of the water and the duration of the stay are not so important as the age. He says also that up to 150 feet there is not much inconvenience, but that every two or three feet after that makes a difference. When diving at great depths they have always a sense of fulness in the head, buzzing in the ears, and flashes of light before the eyes. This condition is relieved by filling the mouth with saliva and swallowing it. Probably this is partly due to the tension in the tympanum being altered by the act of swallowing. On coming up, there is often bleeding from the mouth, nose, and ears, also pain. The patient also referred to the condition which he speaks of as the "bends," that is, wrist-drop and foot-drop, which he has known occur after working at a depth of eighty feet. This usually lasts from two to twenty-four hours, when it completely passes off, unless it is an exceptionally bad attack. The condition was ascribed by the patient to the bands applied round the ankles and wrists to make the diving suit water-tight. A patient may sometimes go two or three months without an attack, sometimes he has two or three a week. This patient has been troubled with "the bends" for fourteen months, and apparently it is a condition which may be entirely separated from the condition which we are dealing with—divers' paralysis. You will note that the disease as we saw it was simply a very mild paraplegia, with imperfect loss of sensation and some subjective sensations in the soles of the feet, and very little else. But as a fact, if we look up the cases which have occurred and the accounts which have been written of this disease, we find that the paralysis from which divers suffer may be exceedingly variable, that it may, indeed, be of almost any degree of severity, from a weakness lasting a few hours to a weakness which becomes permanent; or it may cause an illness which only ends in death. Therefore you have here some pathological condition of the spinal cord, which may be very slight or very severe; and it may be severe in two ways, that is, so severe as to be permanent, or so severe as to terminate life quickly. It appears that in

the majority of cases the symptoms are only as slight as in this man. In the previous attacks which our patient has had he has got well in from three to ten weeks. We have reason to believe that he will resume work after his present illness in a comparatively short time.

The localisation and character of the symptoms are not specially distinctive. In this case if we had not had a history of the diver's occupation and the way in which the paralysis came on, we should have been only able to look upon it as a case of a comparatively mild form of paralysis of the lower extremities due to a slight lesion in the spinal cord, which might or might not be recovered from, but of which we should not have been able to say what was the origin. There is nothing to distinguish such a case from ordinary cases of myelitis. As a rule the onset of the disease is sudden, immediately related to the obvious cause. The patient goes down to great depths and is subjected to considerable pressure. The pressure which exists under these circumstances is known to be three or four times that of the atmosphere. It has been recognised as an obvious fact that paralysis is much less likely to take place if divers are drawn up slowly and quietly than if they are brought to the surface comparatively quickly. You will remember that on the occasion on which the paralysis in this man started he was drawn up in one minute from a depth of about 160 feet—about one-fifth of the time usually occupied. Sometimes the symptoms come on immediately the patient reaches the surface; at other times a few minutes elapse. Perhaps the patient first feels numbness in his legs and a sense of weakness. In severe cases there is paralysis of the sphincters, and other evidence that the spinal cord is involved, and there is more or less complete failure of the functions of the lower half of the spinal cord. Now there is not much difficulty in the diagnosis of such a case. The diagnosis depends upon the associated circumstances. And in regard to prognosis one has to determine that by the severity of the symptoms at the time.

We next come to a most important and most interesting part of the subject, namely, the pathology of the disease. It is obvious that the pathology of the disease is intimately related to the change of pressure which the patient undergoes, that is, change from normal pressure to greatly increased pressure, and then back again to normal.

The latter of these stages is that which determines the symptoms. The explanations of the disease are those which accord with that view of the question. I may say at once that the opinion generally held now is that the solution probably lies in the likelihood that the gas has been forced by the pressure into the blood, and that therefore the blood current holds more gas than it normally should, and that when the patient is drawn up to the surface the pressure being relaxed the air escapes from the blood and mechanically presses upon and interferes with the structures of the spinal cord. It has been shown experimentally that such escape of gas can take place. The pathological results of these cases are not very numerous, because there have not been many cases examined which have succumbed to this disease. But it can be stated that as a rule hæmorrhages are not present, and hæmorrhage is obviously not an explanation which suits all cases, though it may be present sometimes. Then, a certain amount of destruction and disintegration of the cord has been found in some cases, and in a case published by Professor von Leyden he said he found fissures in the spinal cord which were occupied by leucocytes. But from their appearance it did not seem probable that the leucocytes had made fissures, but that the fissures had been made beforehand, and the leucocytes had subsequently occupied them. The conditions seen are obviously unusual, and not what one would commonly see as a result of leucocytal infiltration. One interesting question is this:—why should these lesions take place in the lower end of the spinal cord so frequently as they do? What usually happens is that the lower extremities are affected, possibly the sphincters as well, but the upper extremities commonly escape. Therefore, whatever the lesion is it must be situated below the cervical enlargement, either in the lumbar or dorsal region, and in the cases in which the lesion has been seen it has been in the dorsal region. One cannot leave this subject without referring to an ingenious hypothesis that was put forward by the late Dr. Moxon in some lectures he delivered in 1881 before the College of Physicians—the Croonian Lectures—the subject of which was the influence of the circulation upon the nervous functions. He dealt with the various conditions of the cerebro-spinal fluid, with the condition popularly

known as "congestion of the brain," and some other affections. Among these he called attention especially to the peculiar symptoms and conditions which follow upon rapid removal from greater to less depths in the case of divers. The point which he tried to make was briefly this:—He showed the very defective circulation, as he considered it, of the lower part of the spinal cord; he pointed out that the spinal arteries (one anterior and two posterior) start from the vertebral arteries and run the whole length of the spinal cord, having a diameter almost the same through the whole distance, and that they are reinforced by various vessels which come along the roots of the nerves through the intervertebral foramina. He pointed out further that the nerve-roots in the cervical enlargement go out almost transversely; but, as you go lower down, the nerve-roots take a more and more oblique course, so that in the cauda equina, the last sacral nerves arise from the tip of the spinal cord opposite the lower border of the first lumbar vertebra, and have to go out through the sacral foramina, several inches lower down. Now the reinforcing arteries come through these foramina, and the reinforcing arteries which come along the nerves of the cauda equina have a long journey before they reach the vessels which they reinforce, and the part of the cord which they help to supply. Dr. Moxon inferred, therefore, that so far as the circulation was concerned this was a part of the spinal cord which was unfortunately situated. He got some confirmation of that view from the late Dr. Carrington, who found by carefully looking at the subjects in the dissecting room that the lower spinal arteries were not filled with the injection, although the body was injected from the femoral artery and not from the aorta. That was an interesting point to make. Now it is right to say that Dr. Moxon simply based his explanation in divers' paralysis on the probability that if there was a deficient supply or difficulty in the way of the blood getting to and from this part of the spinal cord, it would be exceptionally liable to suffer from any sudden changes of pressure whatever, and therefore if the patient had undergone great pressure by being immersed in the ocean, when he was suddenly brought to the upper air this was the part most likely to suffer. I do not see any reference to the possibility of the symptoms being due to the extravasation of gas or the

escape of gas from the blood-vessels ; Dr. Moxon seems to have looked upon it only as a matter of blood pressure.

How it really occurs still remains somewhat doubtful, and it cannot be absolutely demonstrated post mortem, because it is not likely that after death you will find the gas bubbles there. What seems conformable to the idea that it is gas is that very often recovery takes place within a short time. Gas may be again rapidly absorbed ; but if it is extravasated in very great quantity it may, before its absorption, do much mechanical damage. It is obvious that in cases which last a very long time the damage done must be more or less permanent in character, and it is quite conceivable and probable that changes of the nature of inflammation or myelitis may be set up by the original mechanical irritation by the gas which escapes from the blood within the vessels, or may even pass outside them.

The treatment of such a case resolves itself very much into the treatment of early myelitis in the acute form, not, indeed, by violent antiphlogistic methods such as blisters and the cautery, but by rest in the early stages, by galvanism and massage in later stages, and care of the bladder at all times. On the former occasions in which this patient has been in this hospital for the disease he has not required vigorous measures.

I have now to show you a case of a disease, also nervous, which is met with more frequently, namely one of lead palsy. The patient is a man fifty-four years of age, and was admitted for loss of power in both upper extremities. He has previously enjoyed good health, and had no disease of importance. He is said not to have had syphilis. During the last year he has worked in an iron-monger's business, and has had to deal both with red-lead and with white-lead ; but he has always taken the prescribed precautions to avoid inoculation with these substances. He is not a teetotaler, but has never drunk to excess. His present disease commenced on the 22nd of October, when he began to suffer from colic and constipation. He was treated ; and as the lead colic got well he began to notice, in the beginning of December, a weakness in the upper extremities, starting in the shoulders and accompanied by some pain and weakness extending

down the arms and forearms. He also had weakness of the legs, so that his walking powers have not been so good lately. He occasionally suffers from palpitation of the heart. You will notice that his arms hang by his side in a helpless manner, and the only way in which he can move them is by lateral movements of his shoulders. You will notice that either shoulder stands out at a sharp angle, due to the great atrophy of the deltoid ; there is not the roundness which is normal to this part of the body, and you can see a depression between the acromion and the head of the humerus. If you ask him to perform any of the movements for which the deltoid is responsible he fails. You will notice the very blue condition of his hands and forearms ; this is largely due to the facts that the hands are constantly dependent, and that his muscles are not contracted, causing the circulation in the part to be defective. If we tell him to raise his hands you will see that in trying to do so he contracts the supinator longus and the pectoralis muscles, there are twitchings in his biceps and triceps, but there is no real action of the extensors ; sometimes he gets a few contractions of the flexor muscles. On the other hand, there is a point here which is constantly noticed in lead paralysis—that if one supports the first phalanges, he is able to both extend and flex the second and third phalanges. His triceps muscle is obvious, but it is wasted. You of course know of the association of extensor paralysis or wrist-drop with lead poisoning. This man presents some differences from the ordinary type. A great number of subjects of lead palsy come to you and hold out their hands in a position of pronation, and say that they cannot do any more ; but this patient for obvious reasons cannot even do that, on account of the state of his deltoid. You may ask what proof I have that it is lead poisoning ; and I shall then point to the history, to the clinical features of the paralysis, and to the fact that he has a distinct but fine bluish-black line along the edge of the gum adjacent to some of the teeth.

It is, indeed, a case in which, besides the extensor paralysis which is so typical of lead palsy, we have some other muscles involved, especially the deltoid, the supinator longus, the brachialis anticus, and the biceps.

This conforms with what has been already often

observed, *i. e.* that while the most common affection is one of the extensors, sparing, however, the supinator longus and the extensor ossis metacarpi pollicis, the next most frequently to be involved is the deltoid, in company with the biceps, the brachialis anticus, and the supinator longus. Knowing that fact one must have no difficulty in regarding this case as one of lead paralysis, because the affection of the deltoid is nearly as characteristic of lead poisoning as the affection of muscles supplied by the musculo-spiral. There are other cases in which you get the small muscles of the hand affected, such as the hypothenar eminences and the interossei; in this case those particular muscles are practically spared. There are other and much rarer cases in which the legs are affected. This man has some slight affection of his lower extremities. If he sits in a low chair he cannot rise again without assistance. Often the muscles affected in the lower extremities correspond to those involved in the upper. There are other cases of lead palsy in which the paralysis is much more extensive, a general affection of the muscles of the body.

There is another feature of the case I should have liked to show you in detail had there been time, namely, the galvanic and faradic reactions. Lead palsy as a rule shows in a very typical form the so-called reaction of degeneration. If you take a case like this and apply the faradic (interrupted) current to the extensor muscles, you will get in them no contraction whatever. If you have a strong current and apply it to the extensors you commonly find the flexors act by conduction through the tissues. If you have one pole on the neck and the other pole on the extensors of the forearm, there is contraction of the muscles lying between, while the muscle to which the pole is directly applied fails to contract. I will show you from the observations of the clinical clerk, Mr. P. Turner, the reactions to faradism and galvanism. The deltoid, brachialis anticus, supinator longus, and extensor muscles react badly to the faradic current or not at all; the trapezius, biceps, and opponens pollicis react slightly; the triceps, pectoralis major, and flexors of the forearm react normally. With the galvanic current, in the deltoid $ACC > KCC$, in the extensors of the forearm, and in the opponens pollicis $ACC = KCC$, in the supinator longus, trapezius, biceps, triceps, pectoralis

major, and flexors of the forearm $KCC > ACC$, which is, as you know, the normal reaction.

Thus, broadly, there is correspondence between the paralysed and atrophied condition of the muscles on the one hand, and their inadequate contraction to the faradic current, and the change in the polar reactions, KCC and ACC, on the other hand.

Time does not permit me to go further into interesting points connected with lead poisoning. I will merely say here that the treatment appropriate to a case of this kind is (1) avoidance of the cause of the impregnation with lead; (2) the systematic use of the continuous galvanic current to the affected muscles; and (3) the internal administration of iodide of potassium, which appears to assist in the elimination of the metallic poison from the system.

Neave's Food.—The various reports from experts on this genuine food agree in saying that it is well adapted for infants, invalids, and persons of an advanced age. All the elements of food are present in an easily digested form, and the flesh-forming ingredients are abundant, whilst the large percentage of fat-producing materials is an effectual contribution towards the maintenance of the heat and work of the animal mechanism. Its highly nutritious properties and careful preparation render this food peculiarly valuable.

Byno-Phosphates.—A malted chemical food (Allen and Hanbury). This excellent preparation is without doubt a most important advance in therapeutical agents. This malted chemical food is a valuable variant of the well-known Parrish's food, in which the active malt extract Bynin takes the place of the inert sugar of the usual preparations. This modification contains in solution the phosphates of iron, lime, potash, and soda, and presents the additional advantage of digestive aid by reason of the nutrient and digestive constituents of the liquid malt. In the treatment of difficult cases of delicate children and patients of impaired constitution this admirable combination will be found to possess distinct advantages.

THE SURGICAL AFFECTIONS OF THE STOMACH, AND THEIR TREATMENT.

A Course of Lectures delivered at University College during February, 1898, by

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No. II.

Now, looking at the list of operations which I have put before you, you will be struck by the fact that all the earlier ones were designed to save or relieve patients suffering from cancer of the stomach, or, more accurately, of the pylorus. But gradually the basis and scope of the operation widened, as it was found that the stomach was tolerant of interference; and, as skill increased, large portions of the organ began to be removed for various growths, or even the whole of the stomach was removed without destroying the digestive functions. This has been done several times, and three or four years ago, when I was in Berlin at Easter, a friend of mine, Dr. Schuckhardt, of Stettin, showed me a photograph of at least four-fifths of the stomach which he had removed in the preceding February, and the patient was still alive and well. It has been done recently in America and Zurich I think. I am not advocating this procedure here, and I hardly think anyone in England is likely to do so. Finally, non-malignant conditions have been brought within the range of surgical treatment, and these bid fair to exceed those treated for malignant disease, both in number and success.

But let us first confine ourselves to the consideration of the operative treatment of new growths of the stomach. And, in the first place, what are they? Undoubtedly the most common is carcinoma. Sarcoma has been found, and more recently we have had large myo-fibromata removed. I present here a drawing of such a large fibro-myoma removed successfully by my friend Prof. Eiselsberg, of Königsberg (Fig. 20). Next I give you here a list which was compiled by Dr. Haberkant, of Dantzig, showing the nature of the disease for which pylorotomy was performed in

forty-four cases, and arranging the growths under certain heads, which will give you an idea of the relative frequency of the various forms of cancer

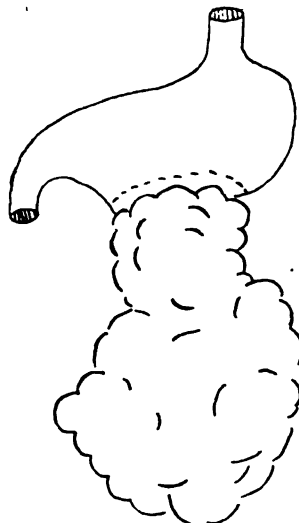


Fig. 20.—v. Eiselsberg's fibro-myoma of stomach.

of the stomach, as microscopically examined after operation. You will find here forty-four cases verified by microscopical examination.

TABLE II (from Haberkant).

Diseases for which Pylorotomy was done in forty-four cases verified microscopically.

Growth	Number of Cases.	Recovered.	Died.
Scirrhus...	16	10	6
Adeno-carcinoma	10	5	5
Soft cancer...	9	1	8
Colloid cancer...	9	7	2
	44	23	21

From Mr. Goffe's collection of cases I have had a table (IIa) arranged in the same way as far as possible.

You will see from these tables the order of frequency of the various growths of the pylorus, and also that operations for scirrhus were the most successful, those for adeno-carcinoma were next in success, the result in soft cancer is very hopeless. As to the most frequent seat of these neoplasms, there can be little doubt that they are, beyond all question, most prevalent in the pylorus; the next commonest situation is the cardiac end of

the stomach, then the anterior part. I have myself operated on two cases in the anterior wall of the stomach. The reasons for the frequency of pyloric cancer are the subject of much speculation at the present moment, but they need not be fol-

Table IIa (Mr. Goffe's tables).

Diseases for which Pylorectomy was done in forty-four cases—English and American.

Growth.	Number of Cases.	Recovered.	Died.
Scirrhus.....	4	1	3
Carcinoma	32	18	14
Adeno-carcinoma	1	1	—
Colloid	2	—	2
Sarcoma	2	1	1
Cicatricial	1	—	1
? Malignant	2	2	—
	44	23	21

lowed out very closely in this surgical sketch. But this much may be said, that those morbid conditions which lead to the prevalence of pyloric ulcer and stenosis probably influence the appearance of cancer in this region, and that there are many who hold that pyloric cancer has, in many cases, its starting-point in chronic ulcer of the part. Hauser, writing in 1883 on this question, after a careful analysis of cases of simple round ulcer of the stomach, as they are called, states that his observations led him to the belief that five or six per cent. of such ulcers of the stomach or their scars ultimately developed into carcinoma. This may or may not be true, but it is highly probable, and if believed in must materially influence our treatment of chronic ulcerative conditions. The symptoms produced by cancer of the stomach are only characteristic when the pylorus is involved. If the growth be on the anterior or posterior wall alone, apart from the presence of tumour in the epigastrium and progressive wasting, there is little to guide us, and it is often very difficult to locate the tumour. But when it is in the pylorus there is not much doubt. There is constipation, weakness, dilatation, gnawing pain, the tumour can be felt, and the patient occasionally vomits blood.

Now in any given case where we believe the stomach to be affected with cancer we have to

make up our minds as to what course to pursue, and here let me say once for all that we ought to make up our minds without delay. Any postponement in coming to a conclusion can only be dangerous, whether we choose this or that operation; whatever we do should be done at once. In deciding for or against operation, the considerations which will weigh most with us are these:—the object of the operation, the possibility of cure without recurrence, the duration of life if recurrence takes place, and then the amount of relief of suffering which is obtained from such a measure, whether such recurrence is going to take place or not. At the present day we possess a good deal of evidence on all these points, based on the experience of large numbers of operators. And let me say here that the experience of one surgeon who has operated on many cases, or of two or three who have each done a number of cases on the same lines, is infinitely more valuable than taking the whole literature of the subject and trying to draw conclusions from the cases therein narrated. It is proverbial that the successful operations are published, while there is at least not the same anxiety to put before the profession the unsuccessful ones. It is only when we have the

TABLE III (from Haberkant).

Results of Pylorectomy published by various operators.

Year of Publication.	Operator.	Total Mortality.	Mortality of Cancer Cases.	Mortality for Non-malignant Cases.
1890	Billroth ...	53·6	57·1	50·0
1890	Lauenstein	66·6		
1892	v. Heineke	64·6		
1893	Lobker ...	46·1		
1893	Schede ...	46·1		
1894	Czerny ...	35·0		
1894	Kappeler ...	35·7	38·1	
Circa ...		50·0%		

complete results of a man who publishes both successful and unsuccessful cases in his series that we have anything reliable to go upon.

For cancer of the stomach we have a choice between pylorectomy and gastro enterostomy, or a procedure made up of both combined, and it is

impossible to say certainly until the abdomen is opened which operation can be done, if any.

But it is not a serious matter to open the abdomen, and this part of the procedure may be undertaken now-a-days without fear. Let us now see what the results of resection of the pylorus have been. I put before you an epitomised table of statistics from 1890 to 1894, comprising the results to hand of a good many skilful operators. You will see that up to 1890 Billroth had a mortality of 53 per cent., *i.e.* in cancer cases 57 per cent.; in non-malignant cases 50 per cent. Lauenstein had a mortality of 66 per cent. And besides this you will note that as time goes on the mortality decreases. Out of a number of cases collected from the general literature of the subject by Haberkant the figures are not very different, though I believe they are less reliable. He examined the records of 359 cases, and tabulated them thus:

TABLE IV (from Haberkant).

Results of Pylorectomy from 359 cases collected from Literature.

Total.	R.	D.	Cancer.		Non-malignant.	
			R.	D.	R.	D.
359	176	183	117	140	30	20

Total mortality 50·9 %

For cancer cases 54 %

For non-malignant cases 40 %

This table is a very fair illustration of the fact that cases collected promiscuously from journals show a lower mortality than the others, the aggregate being 50 per cent., while in the previous tables, showing the total results of certain most skilful and experienced operators, the aggregate death rate was 50 per cent., while there was nothing below 35 per cent., the reason probably being that in the records of particular operators, taken throughout their clinic, there are placed all the unsuccessful as well as successful cases—a condition of affairs which is most probably, if not certainly, not reproduced in the cases recorded in literature.

Another table (IVa) which Mr. Goffe has kindly compiled for me from all the published pylorectomies operated on by American and English surgeons indicates the same thing.

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Table IVa (Goffe).

Results of Pylorectomy from forty-four published Cases—English and American.

Total.	Rec.	Died.	Cancer.		Non-malignant.	
			Rec.	Died.	Rec.	Died.
44	23	21	19	19	2	1

Total mortality ... 47·7 %

„ „ for cancer cases ... 50·0 %

„ „ „ non-malignant cases... 33·3 %

I have already mentioned that out of a number of cases collected from various publications, amounting to 359, the results of pylorectomy are not very different on the whole, though more hopeful, from those obtained by Billroth and others. From Table IV which is based on a large number of cases, you see that the total mortality in it is 50·9 as against 54 of the combined results of a few well-known operations. For cancer cases 54 per cent., and for non-malignant cases 40 per cent.

I have thought it desirable to note here, and this must be borne in mind, that the mortality from this operation for non-malignant disease is shown to be very much lower than for malignant disease, and we shall see this more forcibly when we come to speak of special operations on the stomach for non-malignant disease, the most important branch of our subject.

Another point for emphasis here is, that if we take the first seven years of these statistics (Table V) it

TABLE V (from Haberkant).

To show the Improvement in the Results of Pylorectomy.

1881 to 1887.	Died 64·4 %	Cancer cases.
1887 to 1894.	„ 42·8 %	„ „
1881 to 1887.	„ 42·8 %	Non-malignant cases.
1887 to 1894.	„ 27·7 %	„ „

will be clearer than ever that a great improvement has taken place. You see this by contrasting the seven years' period from 1881 to 1887 with the seven years' interval from 1887 to 1894. In the cancer cases the mortality from pylorectomy was, in the first period, 64·4 per cent., and in the second

period 42·8 per cent.—a very material gain. For non-malignant cases for the first period 42·8 per cent., and for the later 27·7 per cent. Mr. Goffe's figures show the same improvement

Table Va (Goffe).

Improvement in Results of Pylorectomy—English and American Cases.

1882 to 1890 ...	died 76·5% cancer cases.
1890 to 1898 ...	„ 28·6% „

It is clear from all this that the mortality from pylorectomy is still exceedingly high, even in the hands of the most experienced operators in the

mortality from pylorectomy was 47·2 per cent.; that is, 47·2 per cent. of those who died succumbed to peritonitis, 13·1 per cent. died of asthenia, 4 per cent. of shock, 4·3 per cent. of intercurrent disease, and 2·2 per cent. of various diseases. I have had Mr. Goffe's collection tabulated in the same way (Table VI a).

These interesting facts are of importance, and justify some hopefulness, because there can be no doubt that we ought to be able very easily, by improvements in antiseptics or asepsis, to reduce the deaths from peritonitis. Whether we could ever reduce the number of cases that die of asthenia is doubtful, but it is very probable that by a judicious selection that figure too might be

TABLE VI (from Haberkant).

Causes of Death in 165 Cases of Operation on the Stomach, verified post-mortem.

Operation.	Peritonitis.		Asthenia.		Shock.		Intercurrent Diseases.		Various.	
	D.	Mortality.	D.	Mortality.	D.	Mortality.	D.	Mortality.	D.	Mortality.
Pylorectomy	43	47·2%	12	13·1%	30	4%	4	4·3%	2	2·2%
Gastro-enterostomy ..	14	20·8%	15	22·3%	13	19·2%	13	19·2%	12	17·8%
Pyloroplasty	2	28·5%	2	28·5%	1	14·2%	2	28·8%		
	59		29		44		19		14	

Table VIa (Goffe).

Causes of Death in ninety-five Cases of Operation from English and American sources.

Operation.	Peritonitis.		Asthenia.		Shock.		Intercurrent Diseases.		Various.	
	Died.	Mort. %.	Died.	Mort. %.	Died.	Mort. %.	Died.	Mort. %.	Died.	Mort. %.
Pylorectomy	4	19·0	6	28·5	8	38·0	2	9·5	1	4·8
Gastro-enterostomy	11	14·8	33	44·8	12	16·2	6	8·1	12	16·2
	15		39		20		8		13	

world. The reasons for this mortality come out from the next table (Table VI) which I show you. In this Haberkant tabulates the causes of death in 165 fatal cases, verified by post-mortem examination. He has put in the same list a similar analysis for two other operations, but I will ask you to direct your attention only to the first. You will see that he places these fatal cases under the headings peritonitis, asthenia, shock, intercurrent, and various. Under the heading of peritonitis the

lowered very considerably. The same remark applies to shock. Intercurrent diseases, such as hæmorrhage, I am afraid we are not very likely to reduce much, unless it be that asepsis of a more stringent kind will make the liability to bleeding from the opening of vessels by ulceration less than at present. Therefore we may reasonably hope that under practically all these headings still more improvement will take place as time goes on in the results of pylorectomy. But in addition to

the risks of pylorotomy which I have pointed out, you must remember that contraction of the opening may take place, and that, of course, would have to be considered as one of the ultimate dangers following the operation. Such contraction has been known to occur in the further course of the case in several instances.

The next consideration is the duration of life in those who have survived pylorotomy, and the amount of comfort they have had under the new condition of things. The first is fairly represented in these tables which I show you now (Tables VII and VIIa). Here we have an analysis of those cases which survived the operation, and whose future is known.

TABLE VII (from Haberkant).

Duration of Life after Pylorotomy in 51 known Cases.

Cases.			Alive, 1896. Cases.		
Up to 4 months	5	...	Up to 4 months	3	
" 8 "	9	...	" 8 "	3	
" 12 "	3	...	" 12 "	2	
" 2 years	6	.	" 2 years	4	
" 3 "	1	...	" 3 "	5	
" 4 "	1	...	" 4 "	2	
" 6 "	1	...	" 5 "	1	
			" 8 "	1	
—			—		
26			21		

In four more cases alive recurrence was clearly present.

Table VIIa (Goffe).

Duration of Life after Pylorotomy for Cancer—nineteen Cases—English and American sources.

Cases.		At time result was published.		Cases.	
Lived to 7th week	1	Alive after 2 weeks	1		
" 4th month	1	" 1 month	1		
" 15th "	1	" 2 "	1		
" 3½ years	1	" 4 "	4		
	—	" 5 "	1		
	4	" 6 "	1		
		" 7 "	1		
		" 9 "	1		
		" 10 "	1		
		" 15 "	1		
		" 4 years	1		
		" 5½ "	1		
			—		
			15		

These tables give us as accurate information as it is possible to obtain about these patients at present. The amount of relief given in each case was not always the same in those who survived. Some were able to eat anything they liked; others were very far from this, and that is a point we must never forget. The condition of the majority of those who survive may be taken, however, on the whole to have been fairly satisfactory. It must also be remembered, in connection with this list, that since it was compiled some of the cases have added very considerably to their length of survival. Many of the patients appeared to have taken a new lease of life, and to have been able to digest ordinary food in the same way as healthy people. The same is often seen after gastro-enterostomy—a fact of importance, as seeming to indicate that the chief distress from carcinoma pylori is due to the mal-assimilation dependent on the obstruction to the passage of food through the pylorus, and of its consequent decomposition in the stomach. Once the food is allowed to pass on, the forces of the system revive, even though recurrence may be present. Should the new pyloric opening or that provided by gastro-enterostomy contract again, as has been known, the whole train of symptoms and the marasmus return, even before any impression is made on the system by the re-development of the growth.

Gastro-enterostomy.

We now come to the consideration of the reasons for and the causes of death in gastro-enterostomy, as far as we can learn them from published cases. This operation is done, as I have indicated, both for cancerous stenosis of the pylorus, and for other forms of obstruction of the latter, the consequences of which I shall have to allude to more at length later on. I will not put down the figures relating to this procedure under the headings of the various operators, as this would be inconvenient for reasons I need not go into. I will give you them ranged in a series of years. Taking the same fourteen years as before, we have 298 cases, which we will examine (Table VIII). From 1881 to 1887 there are 57 cases, and of these 33 died, a mortality of 55.9 per cent. Later on, from 1887 to 1894, there are a very much larger number of cases, namely, 239, of whom 94 died, the mortality falling to 39.3 for this

period. This larger number of cases and the lower mortality is interesting, because it not only means that this operation is coming more into favour, but that it is being done better. Gastro-enterostomy is, indeed, becoming a far commoner operation, and pylorotomy much less common. These statistics are taken from the work of those who are familiar with both operations, and who have really given us what we know about them. It is quite clear from them and from others that surgeons are more cautious about performing pylorotomy, and that they substitute gastro-enterostomy in many cases which would formerly have been submitted to the more severe procedure. The total mortality over the fourteen years is 42.6 per cent.; that is, 47.5 per cent. for cancer, 25 per cent. for non-malignant affections—a very considerable improvement.

TABLE VIII (from Haberkant).

Mortality after Gastro-enterostomy from Records of 298 Cases.

	Cases.	Died.	Mortality.
1881 to 1887 ...	57	33	55.9%
1887 to 1894 ...	239	94	39.3%
Total mortality for 14 years ...			42.6
" " cancer ...			47.5
" " non-malignant ...			25.0

Table VIIIa (Goffe).

Mortality of Gastro-enterostomy from records of 194 Cases—English and American sources.

	Cases.	Died.	Mortality.
1882 to 1890 ...	45	21	46.6%
1890 to 1898 ...	136	53	39.0%
Total mortality for 16 years ...			42.8
" " cancer ...			41.5
" " non-malignant ...			36.3

We have next to consider the duration of life in gastro-enterostomy, and to contrast that with what obtains in pylorotomy. This comes out in the next table I show you, and it is very interesting. Of those 58 cases of which records were accurately kept (Table IX), there died before the second month 9 cases of gastro-enterostomy for cancer, and 7 died before the third month, 6 before the fourth month, 5 before the fifth month, 3 before the seventh month, 1 before the eighth month,

1 before the twelfth month, 10 before two years. In 1896 there were alive—after four months 4, after eight months 7, after twelve months 4, after two years 1.

TABLE IX (from Haberkant).

Duration of Life in 58 Cases of Gastro-enterostomy.

Died before	...	Alive, 1896, after
2. months 9	...	4 months 4
3 " 7	...	8 " 7
4 " 6	...	12 " 4
5 " 5	...	
7 " 3	...	
8 " 1	...	
12 " 1	...	
2 years 10	...	2 years 1
42		16

Table IXa (Goffe).

Duration of life in sixty-two Cases of Gastro-enterostomy for Cancer from English and American Cases.

Died before	Cases.	At time result was published.	Cases.
2nd month ...	15	2 months	3
3rd " ...	4	6 " ...	2
4th " ...	5	9 " ...	3
5th " ...	6	12 " ...	2
6th " ...	5	18 " ...	1
8th " ...	3	2 " ...	1
9th " ...	2	4 " ...	1
10th " ...	2		—
12th " ...	1		13
13th " ...	2		
16th " ...	1		
17th " ...	1		
22nd " ...	1		
25th " ...	1		
49			

The longest survival after gastro-enterostomy appears to have been in a case operated on by Hahn, the patient being without suffering for seven years after. In this case the growth was believed to be carcinoma by every one who had an opportunity of seeing it during the operation. However, doubt has been cast upon that case, and we are tempted to share in that doubt. In what proportion of cases contraction of the opening into the stomach has followed gastroenterostomy it is difficult to say accurately, but many cases are on record of this sequel.

Having now duly considered the risks of pylorotomy and gastro-enterostomy during and after the operation, let us try to ascertain from the material before us which operation, if any, should be chosen in any given case of carcinoma pylori. I think it may be said that the risks of gastro-enterostomy are in a great measure the same as those of pylorotomy, except that the shock is not nearly so severe, owing to the shorter time consumed in the procedure, and that the liability to leakage of the line of suture is less. But there are other risks peculiar to gastro-enterostomy, as we shall see later on. It is clear that in the past too many patients have been dealt with by both methods, cases which were really desperate, that should not have been operated upon by either method. This was almost inevitable until experience had been gained, experience which such tables as you see before you supply. We now know that in very advanced cases of cancer of the pylorus no surgical measure is likely to be followed by benefit to the patient, while the risks are enormous, and such cases are not operated upon now. But with a relatively early diagnosis the case is somewhat different. A patient may obtain considerable relief, and at a smaller risk than formerly. If, then, on opening the abdomen we find a tumour, obviously cancerous, in the pylorus, that it has not yet contracted any adhesions to the liver or the abdominal wall, the gall-bladder, the pancreas, or the colon, and that the glands in the lesser omentum or the great fissure of the liver are not infected, if we find that the growth has not spread too widely in the stomach or the duodenum, the propriety of pylorotomy may, at all events, be entertained if the patient appears able to bear an operation lasting from one hour to six and a half hours (the latter time having been taken in the case of one patient who survived), and the result of the operation may be favourable. Of course the probable duration of life and the amount of relief to be obtained has all to be judged in the light of the statistics we have been considering. But if, on the other hand, after opening the abdomen we find adhesions between the cancerous mass and the adjacent parts, if we find infected glands, or that the growth involves too much of the stomach or duodenum, *if any* operation is done, it should be gastro-enterostomy. This latter operation ought to take less time than pylorotomy, and will probably

relieve the patient as much as pylorotomy, and keep him alive as long as that measure would under the circumstances. As to the time consumed in the two procedures, we have some records which show a marked contrast in the duration of each taken in various cases, although it is much to be regretted that this point has not been noted in all. To me it appears most important that the time spent over every abdominal operation should be known. This table shows the length of time occupied by eighty-nine pylorotomies and sixty-six gastro-enterostomies :

TABLE X (from Haberkant).

Duration of Operation.

PYLOROTOMY.

Hours.		Died.	Recovered.
1 to 2	37	14	23
2 " 4	45	23	22
4 " 5	4	3	1
5 " 6½	3		3
	89		

GASTRO-ENTEROSTOMY.

Hours.		Died.	Recovered.
½ to 1	16	6	10
1 " 2	42	15	27
2 " 3	7	4	3
3 " 4	1		1
	66		

Of the pylorotomy cases you will see that 37 took from one to two hours, 45 from two to four hours, 4 from four to five hours, 3 from five to six and a half hours. You will see the relative mortalities under these circumstances. The gastro-enterostomy cases did not take anything like so long :

16 took ½ to 1 hour.
 42 " 1 " 2 hours.
 7 " 2 " 3 " "
 1 " 3 " 4 " "

I think the last of these was done in St. Mary's Hospital, the operator describing it as having lasted four hours. The first of my own, which I did ten or twelve years ago, lasted one hour and thirty-five minutes, the next forty-seven and a half minutes, the next one and a quarter hours, the next, I think,

forty-five minutes, and the last two fifty and fifty-six minutes respectively. This is some indication of the improvement in this respect which may come about by practice and experience, and it is only what one would expect. But I remember that after I had finished my first operation I felt that I had been an unconscionably long time. It is clear from this table that the time lost is not nearly so great, and the amount of handling and consequent loss of blood is not by any means so severe in the smaller operation as in the greater. A knowledge of all these figures and facts will, of course, greatly influence our choice of the operation to be done, or determine us against interference of any kind. Now, for my own part, no case has ever been placed in my hands in which there could be any question of pylorotomy; they were all too late for that, and some, as it turned out, even for gastro-enterostomy.

In some cases where the patient's general condition is fairly good, and where the glands are not apparently infected or adhesions present, but where the cancerous growth does involve the stomach and duodenum in such a way as to render the approximation of the two tubes impossible after the excision of the cancerous tissue, the latter may nevertheless be excised, and the end of the stomach may be closed by turning in its serous edges and stitching them. Then the same method for the closure of the duodenum is carried out, after which gastro-enterostomy is done, either by the anterior or the posterior method. This combination of pylorotomy and gastro-enterostomy has in some cases given excellent results, as good as any yet obtained; but of course the procedure is necessarily lengthy because you have two operations instead of one, although you may assume that the pylorotomy done in that way, by turning in the ends of the duodenum and of the stomach, should be performed much more rapidly than the operation where the two had to be adjusted carefully together. This operation is rather an old one, but has been recommended afresh recently as typical by so distinguished a man in this particular branch of surgery as Professor Péan of Paris, who in a recent debate gave it as his opinion that this was the procedure *par excellence*.

The causes of death in gastro-enterostomy, in addition to those which we have seen following pylorotomy, are, as I have already hinted, chiefly

due to the imperfect emptying of the stomach by the new opening, or on account of contraction of the latter, or its having been made too low down in the small intestine. Now the imperfect emptying of the stomach in some cases is easily explained, and I have made a little diagram which I think will make a lengthy description of the faulty mechanism unnecessary (Fig. 5a). In these cases where gastro-enterostomy has been done it has been found, although union has been complete (I am speaking of the anterior method), and an adequate opening has been established, that the afferent and efferent limbs of the loop attached to the stomach are working unsatisfactorily. What happens is this:—you may re-

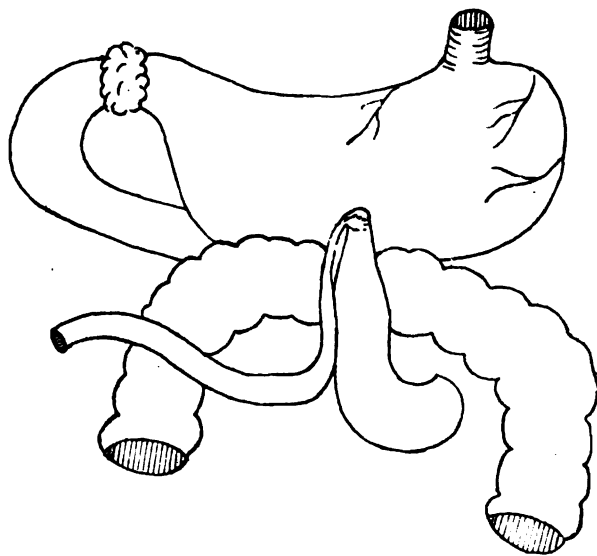


Fig. 5a.—Anterior gastroenterostomy (defective).

member from what I mentioned the other day that when the small intestine is united to the anterior wall of the stomach there is a strain both upon the loop and on the colon (Fig. 5a). The colon and omentum are heavy, and therefore pull the former downwards. Again, the intestine itself drags more or less, and in spite of the fact that you may attach the loop for a considerable distance each side of the opening in the stomach, this dragging brings the two limbs parallel to one another. The consequence is that as the secretions of the liver and pancreas come down the duodenum they fill up this afferent loop. They have, moreover, to rise to reach the stomach, the afferent limb therefore distends, and pressure is

produced upon the efferent loop. Owing to this a spur is produced between the two limbs which directs the bile into the stomach, and prevents its entrance into the efferent limb. The stomach, of course, will sooner or later contract, and will force out its contents; but the tendency is for those contents to be driven into the widest loop, the spur between the two preventing access to the efferent portion. We have, therefore, another reason for this distension, namely, the contraction of the stomach, and the consequence is that a vicious circuit is established, and an obstruction results, which is manifested first by bilious vomiting and ends in death. This accident is very likely to take place in Woelfler's method, and has

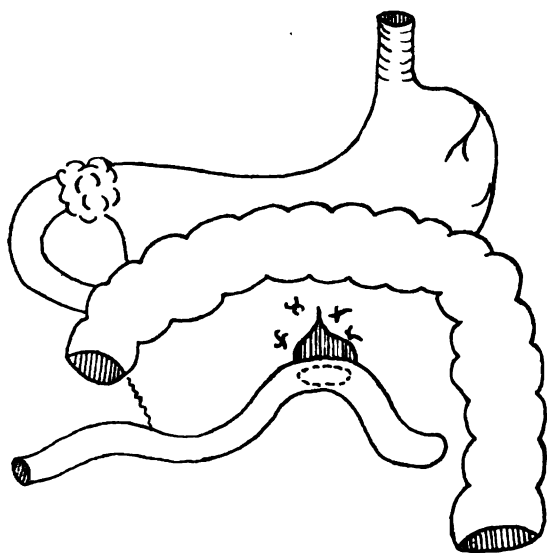


Fig. 6.—v. Hacker's posterior gastro-enterostomy.

occurred to the very best operators. There are several ways to avoid it, and these I will mention to you. It may be avoided by v. Hacker's posterior operation, which I think I described to you at our last meeting. In this, as you see in Fig. 6, the colon and omentum are drawn up over the stomach, and a hole is torn in the mesocolon, and the jejunum attached to the stomach through that rent. Here there is no liability of kinking; there is nothing to contract on to the two loops, and, as a matter of fact although there is regurgitation in many of these cases, it shows an immunity from that accident of obstruction which has been so fatal after the other method. Again, the natural tendency after this operation will be for gravitation

to carry the contents of the stomach into the jejunum. To a certain extent the danger of this accident may be met if, as many have done, the piece of intestine leading up to the opening and that leading from it are stitched for a considerable distance along the stomach after the opening is made. The curve which the bowel then makes is very much greater, consequently there is less liability to kinking. And then there are other ways of avoiding the latter, namely, by either making a valvular opening, as was aimed at by Kocher in his modification of the procedure, the details of which I cannot go into now; or we may do as has been recommended by Braun and Jaboulay,—that is, after making the usual opening you can unite the afferent and efferent limbs of the loop by a second lateral anastomosis as they lie side by side, so that the bile and pancreatic juice can flow direct from one to the other. That seems to be going rather out of the way; but the manoeuvre has been employed successfully, and we must admire the skill, if not always the judgment, of those who have proposed and practised it. It seems to me rather an extensive precaution to take on the off chance of the anterior operation turning out badly.

The way to avoid contraction of the orifice which has been spoken of, is to stitch the mucous membrane of the opening over the edges of the suture which you have made in the serous and muscular coats. Of course for this you require an extra line of sutures, but when the parts are completely covered with mucous membrane there is not the same liability to contraction as otherwise.

Many cases are on record in which very competent operators have by mistake united the lower part of the ileum to the stomach instead of the upper part of the jejunum, and the patient has died of inanition, although the operation has been recovered from perfectly as the union has proved. But the patients have died of starvation, being deprived of the absorption along the whole length of the small intestine. Now a very distinguished operator and writer upon this subject, who has done a great deal to enlarge our knowledge of the whole subject before us, is probably responsible for that in some measure. When surgeons began to be conscious of the difficulty of finding the first part of the jejunum, he suggested that it was certain to be right if you simply pulled up the

transverse colon and omentum, put in your hand under the latter, and took the first coil of intestine to be found, and united that to the stomach. But those who have followed this recommendation have found afterwards, in many cases, though the operation has gone off properly and the patient has recovered, that they have made a mistake. I have read several records recently of this having taken place. Sometimes the ileum has been attached to the stomach not more than a foot or two from the ileo-cæcal valve; of course, such a patient had to starve.

Now a few words about the relative dangers of the anterior and the posterior methods of gastro-enterostomy, a point worth knowing if we are going to select our operation. Of 104 gastro-enterostomies performed by Woelfler's method 45 per cent. died; of 49 performed by von Hacker's 42 per cent. died. There is therefore little difference in the mortality. Moreover, the difference is less when we remember that Woelfler's operation is the oldest, and many of the cases done by his method belong to a period when surgeons were only learning how to avoid dangers which we now recognise, and they had not yet learnt which cases to select for operation. We shall see, however, later on, when we talk of the non-malignant cases, that one surgeon at least, Professor Carle, of Turin, who, so far, has had the best results of any in gastro-enterostomies for non-malignant disease, has almost exclusively employed v. Hacker's method. The latter is undoubtedly the more difficult operation of the two, but it is more desirable as to subsequent function; moreover the results in Carle's cases have not been distanced by any others. The drawbacks to Woelfler's method have been so keenly felt by several operators that many methods, some of which I have described, have been adopted to overcome them, and this in itself indicates that none of them have been found generally reliable.

The question of the prolongation of life is our next consideration. The gain in this respect, it must be confessed, from the two operations of pylorotomy and gastro-enterostomy is not so very great, but apparently the contrast in this respect is in favour of pylorotomy. Out of the 58 cases of gastro-enterostomy, only 12 lived for one year, and out of 47 pylorotomies 22 lived for more than one year. Again, among the gastro-enteros-

tomies, with the exception of Hahn's case, there are none which survived over two years; while among the pylorotomies there are 12 which did survive over two years. All these facts taken together would of course have a certain amount of influence in determining our choice between pylorotomy and gastro-enterostomy. But it may be noted that while the selection of cases and the early recognition of the possibilities of operation would probably influence the statistics of pylorotomy, they would probably have very little effect on those of gastro-enterostomy in this respect. But, however that may be, here in England, the consideration of such facts as we have had before us has usually led to the employment of gastro-enterostomy in preference to pylorotomy. There have been relatively very, very few pylorotomies recorded in this country; this I know from the examination of the best known periodicals which Mr. Goffe has made for me, from which it appears that only 44 pylorotomies performed by English and American surgeons have been published; but there have been a very considerable number of gastro-enterostomies done by English surgeons, and with a fair measure of success.

(To be continued.)

Local Treatment of Articular Rheumatism.

Painting the articulations with methyl salicylate relieves the pain rapidly. Cover with oiled silk and cotton to prevent evaporation; or the following salve can be used:—Liquid vaseline, 20 grammes; methyl salicylate, 12 grammes. If the winter-green odour is disagreeable, salicylic acid can be substituted, although less effective:—Vaseline, 20 grammes; salicylic acid, 4 grammes. Or the combination—Vaseline, 25 grammes; salicylic acid, 4 grammes; sodium salicylate, 3 grammes; extract of belladonna, 1 gramme. Guaiacol has a pronounced antithermic effect, and rapidly soothes the pain. It can be applied pure, but it is best to use one gramme at a time in 5 grammes of 85 per cent. alcohol; or it can be mixed with vaseline:—Vaseline, 25 grammes; guaiacol, 4 grammes. The following combination is very effective, but has a disagreeable odour:—Vaseline, 30 grammes; methyl salicylate, 5 grammes; salicylic acid, 2 grammes; guaiacol, 4 grammes. All of these substances are readily absorbed by the skin.—G. LEMOINE, in the *Nord Méd.*, February 1st.

DISCUSSION ON THE BENEFICIAL EFFECTS OF ONE DISEASE UPON ANOTHER,

At the North-West London Clinical Society, North-West
London Hospital.

Dr. R. H. MILSON in the Chair.

DR. HARRY CAMPBELL, in opening this discussion, exhibited the following table :

I. *Preventive.*

II. *Curative.*

1. Metastasis, counter-irritation.
2. Miscellaneous.
3. Mechanical.
4. Febrile disorders.
 - a. General effects. b. Effects of mental disorders. c. Erysipelas.
 - d. Influenza. e. Vaccination.
5. Traumatism.

He said one disease might act beneficially on another either by preventing it or by curing it. Regarding the prophylactic influence, he might mention the fact that both passive congestion of the lungs and hypertrophous emphysema were said to have a preventive action against tubercle. Then certain germ diseases were preventive of others, e. g. the *Bacillus pyocyaneus* counteracted the activity of the *B. anthracis*.

As to the curative action of one disease on another, that might be considered under various headings, as indicated in the table.

1. Under metastasis we might instance the case of a urethral discharge disappearing upon the occurrence of orchitis. The presumption in such a case was that the orchitis caused the urethral discharge to dry up, and therefore acted in a measure beneficially towards it. Of course the converse might be true, namely, that cessation of the urethral discharge was the primary event, the orchitis being secondary to it. That led them to the consideration of counter-irritation. In counter-irritation they produced one morbid condition in the hope that it might have a curative action on another morbid condition, and in this connection he referred to an article in the *Lancet* in 1842 entitled "The Necessity of Enlightening Public Opinion upon the Subject of Counter-irritation."

2. Under the heading marked "miscellaneous" were grouped cases which could not be referred to other headings. As an instance he might mention a case of paralysis agitans which was cured as far as the tremor was concerned by hemiplegia. The patient was a man who had suffered for years from paralysis agitans, and then developed hemiplegia, and coincident with the latter disease the tremor ceased, and has since remained absent. Another case was one of megrim cured by an attack of acute deafness involving both ears. Another was one in which chronic rheumatism disappeared on the supervention of cancer. He had also notes of two cases of diabetes apparently cured by the occurrence of cancer.

3. Under the heading of "mechanical" benefits might be put the curing of lupus by a syphilitic ulcer, and an interesting case observed by Dr. Green, of Brixton, of a patient suffering from a loose cartilage of the knee; in consequence of an accident to the affected joint traumatic arthritis supervened, and this apparently fixed the cartilage by adhesions, for the man was never again troubled with his knee.

4. The most important class was that in which a febrile disorder seemed to work a beneficial effect upon disease. This subject required discussion under several sub-headings:—(a) First as to the general effects of fever. In a person suffering from fever sugar might disappear from the urine—probably the sugar was burnt off by the hyper-oxidation attendant upon fever. Again, when a person with purulent discharge developed fever the discharge often ceased. Fever might also cause a diminution in chronically enlarged tonsils. (b) Regarding the beneficial effects of fever on mental disorders, it was well known that mental affections were frequently benefited by fever, even permanently. So much was this the case that Dr. Wagner had suggested that patients thus afflicted should be injected with the poison of erysipelas. That seemed to hold out promise of great things in the future. (c) As to the beneficial effect of the poison of erysipelas he would not say much, but hoped that some valuable information would be forthcoming from those present. He would merely mention a case of acute yellow atrophy of the liver, which had greatly improved upon the supervention of erysipelas, a fact which he had not seen referred to in Mr. Mansell Moulin's very able paper in the

'Lancet,' was that erysipelas might cause the disappearance of benign tumours as well as malignant. He (Dr. Campbell) had recently come across a case described by Mr. Hutchinson of a nasal polypus disappearing after an attack of erysipelas; also a case of "pseudo-lipoma," and one in which a mass of callus was absorbed under a similar influence. Such facts seemed to him of great importance in view of the possibility that the absorptive influence of erysipelas on malignant growths might be used as an argument in favour of their parasitic origin, for it might be argued that the poison of erysipelas caused absorption of these growths by destroying their parasites. (d) There was an interesting group of cases in which influenza apparently worked a good effect, and a very interesting case had been related by Dr. Knight, of Brixton. The patient was a lady 45 years of age, who was suffering from malignant disease of the liver. She contracted influenza, which made her very ill, but from that time her liver shrank, and she got apparently quite well. However, the tumour returned in eighteen months, and the patient died. (e) Then there were the good effects due to vaccination. There seemed little doubt that whooping-cough was benefited by vaccination, and so also was chronic eczema.

5. Finally he would allude to the curative effect of serious injury. He was acquainted with the case of a lady who suffered seriously from chronic rheumatism. She met with a severe accident, which necessitated amputation of both her legs fifteen years ago, and ever since then she had been free from rheumatism. An interesting point for discussion was how the accident produced that effect on the rheumatism. Of interest, from a psychological point of view, was the case of an old gentleman who had his foot badly crushed while hunting. Before the accident he was very irritable and unsociable, but ever since he had been quite a model in these respects.

Dr. JAMES TAYLOR thought they would have to deviate a little from the line laid out for them, and consider not only the effect of one disease upon another, but also the alternation of symptoms in the same disease. Such conditions as an access of tubercular process in the lung, after the healing of a tuberculous condition elsewhere, were well known and recognised. He had recently had the opportunity of seeing an old lady who had for many

years suffered from eczema of presumably gouty character. She developed glycosuria, which was present for several months, and during the time that the glycosuria was present the eczema was in abeyance. The glycosuria subsequently disappeared, and then the eczema again asserted itself. That he would be inclined to regard as the alternation of one symptom with another, the eczema and the glycosuria being presumably manifestations of the same gouty condition. The same was true of auditory vertigo. It was well known to several sufferers from this condition, although he did not think it was generally recognised, that people who suffered from deafness associated with occasional attacks of auditory vertigo were inclined at times to welcome an attack of vertigo, because they found from experience that their hearing was materially improved after such an attack. What chiefly interested him in this discussion was the effect of acute disease upon epilepsy. He had on one occasion the opportunity of observing two epileptics, liable not merely to attacks at long intervals, but subject to very frequent fits, suffering from pneumonia in one case and pleurisy in the other. During the time that the pneumonia was present, not only while the febrile temperature was maintained, but for a week or two after the temperature had subsided, the patient, although he had previously been subject to attacks almost daily, remained for certainly three weeks without a single epileptic seizure. The same train of events was noticed in the case of the patient who had pleurisy. Regarding the effects on the mental condition referred to by Dr. Campbell of the injection of toxins such as that of erysipelas, one of the most interesting points he knew in that connection was the effect which was observed a few years ago at the Morningside Asylum, Edinburgh, by Dr. Bruce, of the injection of thyroid extract in cases of melancholia, and of chronic insanity. Dr. Bruce noticed that these patients had sub-normal temperatures for the most part; and he had also observed that some patients suffering from myxedematous insanity had, during the treatment by thyroid, had a very distinct rise in their temperature, and a corresponding increase in their mental capacity, the temperature and the improvement in the mental condition being probably both due to the treatment by thyroid. This had suggested to Dr. Bruce that in those other cases of

melancholic stupor, and other forms of chronic insanity, the artificial raising of the temperature might tend to benefit the mental condition. He treated a number of such cases by the injection of thyroid, and had very remarkable results. Some of the patients improved only temporarily, but others were benefited permanently. Of course in such an experimental therapeutism one did not expect to get absolute cure, or even a very marked alleviative effect in all cases; but in two or three series of cases which were published in the 'Journal of Mental Science' the results were sufficiently important and beneficial to justify a trial of the treatment by thyroid in such cases. Returning to epilepsy, he said that another condition, not a disease, had a very marked effect upon epilepsy,—namely, pregnancy. He had tried, but in vain, to discover some common point in the cases which were free from fits during pregnancy. In some cases the fits ceased entirely during pregnancy, while in others the pregnancy did not seem to make any appreciable difference in the frequency or in the severity of the attacks. In those who were benefited by the pregnancy the fits often recurred during the puerperium, before the patient was able to get about again. He did not propose to theorise, but he thought every one who saw much of disease must have been very much impressed with the undoubted influence of one disease upon another, and, what was to some extent a corollary of that, the alternation of one symptom of a disease with another manifestation of the same disease.

Mr. MANSELL MOULLIN thought the subject of very great interest, and it was one he had been working at for some time. He had come hoping to hear some account of the action of operations on epileptics, and in general paralysis of the insane. His own experience on the curative effect of one disease upon another had been almost entirely limited to the action of erysipelas. He thought there was no doubt that erysipelas had a distinct curative effect upon many diseases, and this was largely recognised in France, so much so that a class of disease was spoken of there as *erysipèle curatif*. Years ago Ricord had published cases in which tertiary syphilides were acted upon by erysipelas and disappeared, and the same thing was true of keloid and lupus, though sometimes the lesions came back again. There was no doubt

that erysipelas acted upon non-malignant growths as well as on growths which were malignant, and therefore he agreed with Dr. Campbell that this was an argument against the probability of malignant growths being parasitic. The most striking effect of erysipelas was on spindle-celled sarcomata, nearly all growths of that nature in Dr. Coley's hands having altered very considerably after injections of the erysipelas toxins. Sometimes the erysipelas had such a marked effect upon carcinomata and sarcomata as to cause the growths to disappear entirely, while at other times the tumour only diminished in size, and recurred after the erysipelas had passed away. The change could not be due to the streptococcus itself, because a similar change was produced by the toxins independently of the streptococcus. The actual microscopic appearances seemed to point towards a very acute fatty degeneration. Some of the records of such treatment state that the tumour became exceedingly soft, and that when an incision was made into it a yellowish-white emulsion, amounting in several cases to some ounces, was let out. This fluid showed under the microscope very few pus corpuscles, and consisted almost entirely of broken-down fatty material. In one case of carcinoma the alveoli were full of broken-down cells mixed up with fatty molecules. The fatty degeneration thus produced must be very acute, because some of the sarcomata had entirely disappeared in three or four days, while disappearance in a fortnight did not appear to be at all uncommon. Acute fatty degeneration could not be simply a question of temperature, for, in reply to a question, Dr. Goodhart had told him that his experience went to show that high temperature in such diseases as specific fevers had no such effect. The change must be due to the products of the erysipelatous organisms. There are a good many analogous arguments which could be used, though the comparisons should not be too close. There was Dr. Beatson's case of oöpheroctomy in recurrent scirrhus of the breast, which caused the scirrhus to disappear entirely; but the experiment had been subsequently repeated with less success, among others by Mr. Stanley Boyd. He knew of one case under the care of a friend in which a recurrent scirrhus entirely disappeared under those circumstances, but he could not say what was the age of the patient—a factor which had a very

important bearing on the matter. As to the action of erysipelas upon callus, he did not know of any case in which it led to the absorption of callus, but he knew of one in which the erysipelas caused an enormous amount of callus to put in an appearance. With regard to the other facts which had been put forward, there were very few upon which he was competent to offer an opinion, but he was not sure that he would regard a case of cessation of urethral discharge and the appearance of orchitis in the light of a cure. He thought it was more easily explained by what Dr. Taylor had spoken of as alternation of one symptom with another. He supposed the conclusion they were forced to was that they knew really very little about the nutrition of the body in health, and still less about it in disease. The thyroid gland had been known for many years, yet it was only very recently that anything definite had been ascertained about it. He did not think erysipelas would ever succeed in curing all malignant growths, but he was firmly convinced that the injection of erysipelas toxins causing a profound alteration in nutrition was an experiment which was worth trying in a great many cases.

Dr. ALEXANDER MORISON thought Dr. Taylor's remark as to the alternation of symptoms held good very largely. Before one could say that a cancer was absolutely cured, the demise of the patient had to be awaited to be sure of the diagnosis of malignancy, and of its non-return. The subject of metastasis was extremely interesting. The metastasis might take place in directions detrimental if not to the original disease or symptom, at least to the organism upon which it existed, *i.e.* the patient. For instance, he had noticed an eczema involving the whole of the head disappear, and meningitis supervene which ended fatally. He referred to a case of a child who was unconscious, with subnormal temperature, and whose illness he thought was some meningitic affection. But in the course of four or five days, though it was in warm weather, an inexplicable bronchitis developed in the child; then the patient began to be more conscious, but he was not sure of his diagnosis until he saw the first faint signs of measles spots gradually struggling out. Notwithstanding the late eruption the patient died. This case showed in a very telling manner the influence of a succession of alternating symptoms upon the vitality of the patient. He thought

the same thing was to be observed in comparatively rare cases of hæmorrhagic smallpox, in which widespread vaso-paralysis with extravasations took the place of the normal eruption. Another instance of metastasis was the very rapid disappearance of marked coryza on the supervention of croupous pneumonia. The influence of fever upon diabetès was well known, whether the fever was continued or symptomatic, and was probably due to some quickened metabolism. He had known the process of vaccination in an elderly woman who was afraid of going to a country in which smallpox was epidemic, give rise in a short time to persistent irregularity and dilatation of the heart, which in a few months' time proved fatal. He had no knowledge of the influence of toxins upon mental disease, but he believed splenic injections had been found to have similarly good effects to those of thyroid.

(To be continued.)

Ophthalmic Crises in Tabes Dorsalis.—

P. K. Pel reports a case of locomotor ataxia with paroxysmal disturbances of both eyes, which consisted of irritation of the sensory, secretory, motor, and vaso-motor nerve-fibres. The most prominent symptoms were severe burning, lancinating pains in both eyes and their immediate neighbourhood. During as well as after the attack a hyperæsthesia of the eyes and their surroundings could be confirmed. The psychical condition of the patient also showed slight changes during the attacks. Pel designates these attacks as tabetic ciliary neuralgias with marked vaso-motor and secretory symptoms, and looks upon them as true ophthalmic crises.—*Wiener klin. Rundschau*, January, 1898; *Medical Record*, April 9th, 1898.

"Frame Food" Jelly.—This jelly is prepared by extraction from wheat bran, and contains soluble phosphates, albuminoids, and other nutritious matter; it possesses the nourishing and digestive properties of malt extract, and has been specially elaborated for the purpose of fulfilling the demand for a modified form of "Frame Food" Extract. As an article of food it is recommended on account of its wholesome and nutritious properties, and its dietetic value is still further enhanced in consequence of the action of the diastase present.

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* Specially reported for *The Clinical Journal*. Revised by the Author.

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A CLINICAL LECTURE

ON CASES OF

TABES DORSALIS, PACHYMEININGITIS SPINALIS, CEREBELLAR TUMOUR, AND OTHER DISORDERS OF THE GAIT.

Delivered at the Hospital for Diseases of the Nervous System, Welbeck Street, January 11th, 1898,

BY

THOMAS D. SAVILL, M.D.Lond., D.P.H.Camb.

GENTLEMEN,—The subject of the disorders of gait will give us the opportunity of studying together certainly the commonest of the chronic affections of the spinal cord, locomotor ataxy, in which disease some very valuable contributions to our knowledge have been made during the past year, contributions which throw a flood of light upon the real origin and nature of the affection. This disease has always been regarded as remarkable for its incurability, but I hope to be able to prove to you that some cases, at any rate, will yield to treatment. We shall also have an opportunity of studying three great principles which govern neuro-pathology. These principles are of prime significance, and have a far wider applicability than the narrow use we shall make of them to-day.

The various conditions in which disordered gait is a symptom may be grouped as follows:

I. Tabes dorsalis, or, as it was formerly called, locomotor ataxy.

II. Other diseases in which the posterior columns are affected, such as (1) spinal tumour affecting the posterior columns, and especially in the lumbar region; (2) posterior spinal pachymeningitis; (3) Friedreich's hereditary ataxy; (4) ataxic paraplegia, or spasmodic tabes dorsalis.

III. Cerebellar disease, and especially cerebellar tumour.

IV. Other affections in which the gait is peculiar,

though disordered gait may not be a leading symptom—diphtheritic paralysis, alcoholic paralysis, paralysis agitans, various forms of lateral sclerosis, hemiplegia, functional or organic, chorea, &c.; and finally—

V. Certain cases of vertigo due to aural, ocular, circulatory, or other maladies; though the disturbance of gait only occurs, under these circumstances, during the attacks, and their consideration may be postponed to a future occasion.

The points to be investigated in any given case where the patient cannot walk properly are as follows.

(1) First, the manner in which the gait is disturbed; what is the abnormality in the gait? Now, there are two typical kinds of disorder in the gait which correspond to the two principal diseases in which the power of walking is altered, and where this is due not so much to any weakness in the legs as to a want of control over them; namely, the ataxy or inco-ordination of tabes dorsalis, and the sinuous or gyratory walk of cerebellar tumour. The former consists of an exaggeration of the normal movements; the patient steps too high, throws out his legs too far, and brings them down too violently on the floor; whereas in the latter the actual steps may be well made, but the patient sways from side to side like a drunken person as he goes along, especially if asked to walk along a straight line. This is impossible for him, and he follows a sinuous or serpentine course, or tends to fall over to one side or the other. In cerebellar tumour, also, the patient may have a tendency to fall forwards or backwards, which in some cases that have been observed indicates the position of the lesion in the upper or lower parts respectively of the middle lobe of the cerebellum. Gilles de la Tourette adopted the ingenious method of blacking the patient's feet and making him walk along a long strip of white paper to record disorders of gait; and possibly this may be useful where a record is necessary.* In the above classification ataxy or inco-ordination is met with in groups I and II, the sinuous or gyratory walk in group III, and various special kinds of defect in group IV.

(2) The second point to note is whether the patient can preserve his equilibrium in a standing posture. This may be lost in both of the affections named,

but far more often in tabes. The other points, which we need not dwell upon in detail now, but which will be referred to as we proceed, are (3) the deep reflexes; (4) whether there is any loss of power or of sensation; (5) the condition of the pupils and of the cranial nerves; (6) the age and sex of the patient, and any other ætiological factors. The age and sex of the patient are most important circumstances, for tabes is practically confined to male patients between the ages of thirty and fifty, whereas cerebellar tumour is most frequent in childhood, though it may occur at any age and in either sex.

1. **TABES DORSALIS**, or locomotor ataxy, may be clinically defined as a chronic disease, commencing with various disturbances of sensation and of the cranial nerves, and terminating in a total abolition of the faculty of co-ordinating the voluntary movements, muscular power usually remaining intact until near the end. This is a purely clinical definition. Anatomically, until quite recently, it was described as a sclerosis of Burdach's columns in the lumbar region, combined with a similar process involving more or less of the posterior spinal roots, the spinal ganglia, and sometimes the posterior horns, and giving rise to an ascending sclerosis in Goll's columns, the antero-lateral tracts, and sometimes the cerebellar tracts above. It is maintained by the best authorities that Burdach's columns in the dorsal and cervical regions are never involved unless the arms reveal ataxy during life. But other parts are also sometimes found to be involved; thus Charcot mentions that the cells in the anterior horns are affected in cases accompanied by joint disease.

This is a brief description of what has hitherto been regarded, ever since the days of Todd, as the essential lesion in locomotor ataxy; but, unfortunately for this statement, there have been at different times cases recorded in which, although the patient revealed all the most typical symptoms of the disease during life, there were no such lesions as those I have mentioned found after death. The posterior columns, nerve-roots, &c., were perfectly healthy.* This difficulty has hitherto been got over by supposing that these observers had overlooked the narrow band of sclerosis, which in the

* 'Nouvelle Iconographie de la Salpêtrière, tome i.'

* A. Hughes Bennett, 'Clin. Soc. Trans.,' vol. xviii; Déjerine, and others.

early stages is only to be found just on the inner side of the posterior grey horns.* But I had the opportunity of examining Dr. Bennett's specimens, and cannot agree with this; and now another and more rational explanation is forthcoming.

During the past year valuable contributions have been made to our knowledge concerning the structures which are subservient to "muscular sense," and about the real lesion in tabes. There is a little structure called the "muscle spindle," which has been known to physiologists for half a century,

these are called the intra-fusal muscle-fibres, and from end to end they divide and reunite longitudinally, so that the number of fibres found in any cross section depends upon the position at which the section is made. Nerve-fibres in great profusion also enter the spindle, and Sherrington* has shown not only that these nerve-fibres are connected with the posterior roots, but that they convey afferent impulses; and there is no doubt, as Kerschner and others maintain, that these are really the end-organs of the muscle sense nerves,

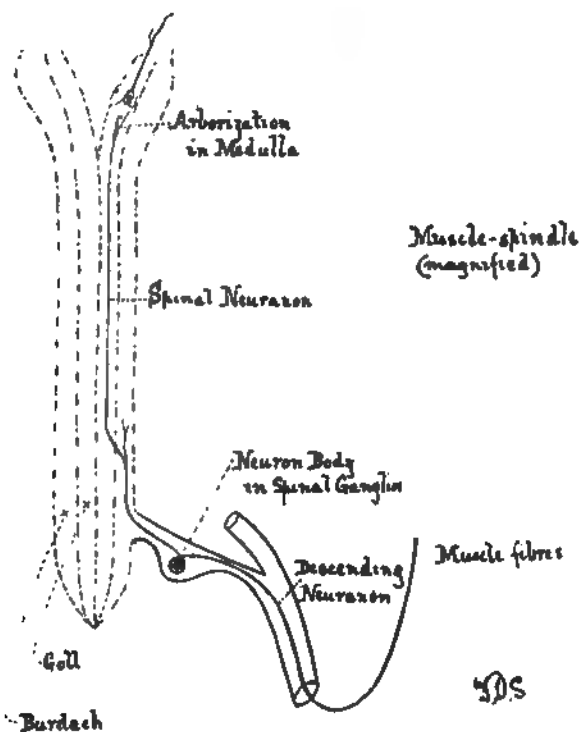


Diagram of spinal cord and muscle sense neuron to show the new pathology of tabes dorsalis. The sites of initial degeneration are at the ends of the descending neuraxon (viz. its termination in the muscle spindle) and the ascending (spinal) neuraxon (viz. its arborization in the medulla).

but which has not had its proper function assigned to it. It is found in all muscles excepting the diaphragm, and the intrinsic muscles of the tongue and those of the eyes. In size they measure about three millimetres by one third of a millimetre. They are fusiform in shape, and consist of a laminated sheath of white fibrous tissue containing within it a lymph space, in which lie small striated fibres resembling muscle fibres in appearance, but about one third of their diameter. Indeed,

and convey to the brain a sense of position of the muscles. Now, it has also been shown by Batten† that these muscle spindles are diseased in cases of tabes, and that they are not affected in the primitive myopathies, progressive muscular atrophy, nor in infantile paralysis. The disorder consists of a degeneration of the intra-fusal nerve endings, and the contiguous muscle-fibres, the nerve fibrilla; and muscle-fibres outside being healthy.

* 'Princ. and Pract. of Med.,' Fagge and Pye-Smith.

* 'Journal of Physiology,' 1896-7.

† 'Brain,' 1897.

You will remember, gentlemen, that the simplest view to take of the nervous system is that it consists of a series of neurons arranged on an upper or cerebro-bulbar level, and a lower or periphero-spinal level. Each neuron consists of a neuron body (the cell) and various processes which terminate in arborisations. One of these processes is covered by a myelin sheath, is longer than the others, and is known as the neuraxon. Now, as a general pathological principle it may be affirmed that *whenever the nutrition of a neuron is impaired, the peripheral endings of its processes are the first to undergo degeneration.*

Applying this principle to the pathology of tabes in the light of Sherrington and Batten's researches, the condition of affairs is as follows (Fig. 1):—The body of the muscle-sense neuron is to be found in the posterior ganglion, and it has an upper or spinal neuraxon, and a lower or peripheral neuraxon; the lower passes down through the posterior root into the spinal nerve, and terminates in the muscle spindle. The upper one passes up the spinal cord, and terminates in relation with the medullary and cerebral cells above. Now, in tabes the end of the lower neuraxon degenerates within the muscle spindle, and we have good reason to believe, though the grounds are not quite so sure as in the case of the lower spindle, that the upper end of the upper neuraxon also degenerates, and we have before us an illustration of the fact that the terminations of a neuraxon are the first to be affected in the degenerative process.

This is a great step, gentlemen, and it helps to explain several things. In the first place, it explains those cases in which no sclerosis of the spinal cord was found, because here at the end of the neuraxon is the commencement,—indeed, the very essence of the disease. In all probability those cases observed by Bennett and others died in an early stage of the disorder, or at any rate before the degenerative process had reached that part of the neuraxon lying in the spinal cord.

In the second place, it helps to explain that feature which has puzzled us so long, namely, why in a spinal affection we should have so many cranial manifestations; and thirdly, it also helps to explain the long pre-ataxic stage, in which the chief manifestations are associated with sensory disturbances, because we may fairly assume that

the other sense neurons of the lower level are similarly affected by the slow degenerative process.

But you will say, how about the sclerosis of the spinal cord, which is known to be present in a large proportion of the cases of tabes dorsalis at the time of death? Now, gentlemen, during the past three or four years pathologists have been much exercised about this question of the increase in the interstitial tissue which occurs in many diseases,—in cirrhosis of the liver, for example, in sclerosis of the spinal cord, and in granular kidney and the like. The point is whether this interstitial fibrosis is a primary change, as hitherto believed, or is it secondary? Finally they have come to the conclusion that in a large proportion of cases this fibrosis or sclerosis is not the primary change, pressing upon and causing atrophy of the parenchyma of the organ, but in fact it constitutes a secondary condition, arising from and replacing the atrophy of the parenchymatous cells. The proofs of this are too long and complicated for me to enter into now, but suffice it to say that the general proposition applies here, and that *the primary change in locomotor ataxy is the degeneration and atrophy of the nerve-fibres, particularly at their ends; and secondarily to this the sclerosis, i. e. the increase in the interstitial tissue, arises.*

Long ago (about 1860) Cohnheim, a pathologist far in advance of his day, wrote this important statement: "The mutual resistance of tissues to each other's encroachment limits tissue growth normally. If you remove one tissue element, by degeneration or otherwise, the surrounding tissues (deprived of this opposition) tend to take on increased activity, growth, and proliferation." This statement was hypothetical at that time, but now it rests on a firmer basis, and the sclerosis in the spinal cord in tabes is really a secondary occurrence both in point of time and causation; and Bennett's cases, which puzzled us so at the time, help to prove it. The first change to occur and the cause of the sclerosis is the degeneration of the muscle-sense neuron; which degeneration starts in the ending or arborisation of the muscle-sense neuraxon,—that part, namely, which is furthest from the neuron-body or nerve-cell, which, as you know, controls the nutrition of the neuraxon, and gradually spreads thence upwards and downwards towards the neuron-body, which itself may

become ultimately involved; and then we get sclerosis of the spinal ganglion.

The *symptomatology* of tabes may be divided into three stages, and it is interesting to notice that in the course of years our knowledge of tabes has gradually, as it were, shifted backwards step by step; so that what was formerly regarded as the real disease is now regarded as its stage of complications. At first it was classed as a paraplegia; now we know that paralysis of the legs only occurs at the termination, and not always then. Next it was regarded as an ataxy; now we are gradually coming to regard this even, like the sclerosis of the cord upon which it probably depends, almost as a complication or sequel; and at the present day we concentrate our attention on what is called the *pre-ataxic* stage; because if we can only recognise the disease in this its early phase there are many cases, and I hope to show you some to-day, in which the disease can be arrested and even cured. I propose, therefore, with your indulgence to study this pre-ataxic stage very closely. Its most important and characteristic symptoms consist of lightning pains, sensory disturbances, and changes in the pupil, with other ocular symptoms.

1. The *lightning pains* of tabes are characterised by being erratic, evanescent, recurrent, and sometimes periodic. Tabes should be suspected in all neuralgias having these characters. Their most frequent seat is the sciatic nerve, but any other nerve may be affected. I would like you to bear in mind in this connection the sharp shooting pains which characterise the early phases of multiple peripheral neuritis, for we now know that tabes in its origin and essence is really a neuritis beginning in the sensory nerve endings. Pains occur also in the situation of various viscera, and come on similarly in attacks. These attacks the French call crises: thus there may be attacks of gastric pain followed (perhaps) by vomiting (gastric crises); or vesical pain, accompanied by retention or incontinence (vesical crises), or rectal pain which may be attended with constipation (rectal crises), and so on. Very frequently the bladder is the organ affected, and, because there is some hesitation in passing water without actual retention, the patient thinks that he has a stricture, a delusion which his doctor not infrequently shares, as in a case I shall show you to-day. Many of these cases, my friend

Mr. Buckston-Browne tells me, in this way come first under the notice of the surgeons who devote themselves to the diseases of the urinary organs. There is simply a delay at starting to make water, and the case may be mistaken for premature enlargement of the prostate. Diminution or increase of the sexual power is not uncommon. Laryngeal crises are occasionally observed, and consist of attacks of laryngeal dyspnoea coming on mostly at night. These are often mistaken for asthmatic attacks, and are very alarming, but the patient recovers in an hour or so. Nevertheless, it is almost sure to return in a few days or weeks; for the features of all of these pains are alike in being erratic, evanescent, and recurrent. The girdle pain is another symptom which is by no means infrequent. In one advanced case which I have recently seen it was present thirteen years ago, though the patient played in a football match five years ago; since when the disease has made rapid strides.

2. Various and remarkable are the many different *anæsthetic* and *analgesic* symptoms observed in this disease, and to describe them fully would be to occupy the whole of the short hour at our disposal. Sometimes there is analgesia without anæsthesia over the whole or a part of the body. The soles of the feet and the ulnar borders of the hands are the favourite situations. Sometimes there is thermal anæsthesia; sometimes delayed sensation; sometimes displaced sensation (*allocheiria*); but the strangest of all, perhaps, is what Duchenne first described as the "*masque tabétique*," in which the patient has anæsthesia in the lips, nose, or face, and it gives him the impression that he has no face. Perversions or abolition of taste or smell are not uncommon.

3. The *ocular symptoms* are very important, and the most frequent of these, perhaps, is transient diplopia, due apparently to transient weakness of one or other of the ocular muscles. My experience quite bears out those who say that quite half the cases of tabes at one time or another suffer from this symptom. The pupils, too, present characteristic signs. The most typical of these is the Argyll-Robertson pupil, a loss of mobility to light but not to accommodation. But it must not be thought that this is the only alteration met with in tabes. I believe myself that the commonest change is an inequality of the two pupils; they

may, however, both be very contracted or both immobile to both forms of stimulus. Another very frequent cranial manifestation is optic atrophy, coming on with a greyish-white appearance of the optic disc, and finally terminating in perfectly white discs with clearly defined edges. Any of these symptoms may precede the ataxy by ten or even more years.

4. Absence of knee-jerk is, in my experience, among the earliest symptoms, though how early it occurs nobody as yet seems quite certain.

5. Various *trophic disturbances* are associated with tabes, thanks to the observations of that prince of clinical observers, Charcot. You will remember that it was he who pointed out that a particular kind of arthritis, now called "Charcot's joint disease," may be present in this malady, and it may occur quite early, at a time when other symptoms are in abeyance. In it a joint becomes swollen, but it is pale and free from pain. Indeed, there is such a complete absence of pain, heat, and redness, the other three classical symptoms of inflammation, that the joint may become quite disorganised or dislocated ere the patient pays much heed to it. In an interesting case narrated by Charcot, the patient was doing his military service and casually found out that he could not march so well as the others. In the knee-joint, which is its favourite situation, the swelling is less likely to be overlooked, but in this case the two hips had gone on to dislocation before the patient was really aware of the disease.* Besides the joints, the nails may be affected with a sort of painless ulceration; or the teeth may be similarly affected and drop out; or perforating ulcers occur in the foot. All of these lesions have the same slow chronic characters.

These are the principal symptoms which, up to the present time, have been associated with the pre-ataxic stage of tabes. You will observe that they are of two categories (*a*) central and (*b*) peripheral, and are no doubt explained by the same kind of degeneration affecting the ends of the trophic and sensory neurons as that which we now know affects the muscle-sense neuron. I have dwelt upon them at some length because it is in this stage that most good can be done. As to their relative value, if the patient be a male, of an age between thirty and fifty, and, in addition,

present symptoms taken from any two of the above groups, I believe that one is justified in diagnosing the disease, for purposes of treatment at any rate.

The ataxic stage.—This consists of the above symptoms, which gradually become emphasised in course of years, combined with (1) the characteristic walk, and (2) the loss of equilibrium on standing, though the power of the muscles remains, and their nutrition is often remarkably good. Indeed, their strength and volume are more often above than below normal. As to the first, the gait, you will see its characters in the patients who are coming in better than I can describe them; but as to the second, the loss of equilibrium on attempting to stand with the eyes shut, which was first described by Romberg, it would be as well to offer you a word in explanation. You know that our equilibrium in the erect posture is preserved chiefly by three means, excluding for the moment the vestibular nerve, which is a nerve rather of orientation than of equilibration. First, by means of sensation as we touch surrounding objects, and especially the sensation in the soles of our feet. Secondly, by means of sight, by which we judge our distance from surrounding objects; and thirdly, by muscle sense, or the sense of the position and state of contraction of our muscles. Now, in tabes the first and third of these are considerably disturbed, and may be totally abolished, and therefore if the patient closes his eyes he is deprived of the only remaining means of preserving his equilibrium. This is the simplest way of explaining this symptom, and recent researches have confirmed its correctness. These patients find a difficulty in starting to walk, but once started they improve as they go on up to a certain point. They always have a difficulty also in turning round, and Fagge mentions an amusing incident of a patient who explained his unpunctual arrival at the hospital one day by his having started in the wrong direction and being compelled to continue until he met with some one who could help him to turn round and start him the other way. In tabes the arms are much less often affected than the legs, at any rate until quite late in the history. The mind also remains clear in most cases until quite the end, and it not infrequently happens that the patient is able to conduct his business for ten, twenty, or even

* 'Nouvelle Iconographie de la Salpêtrière,' tome v.

thirty years after his locomotion has become imperfect, and perhaps after the cranial nerves damaged.

The *terminal* or *paralytic stage* need not detain us long. It may be many years—ten, fifteen, or twenty years—before this stage sets in. It is really a stage of complications, which include very frequently atrophic paralysis, less frequently non-atrophic paralysis; either of which may be slight, or it may be so marked that the patient is unable to leave his bed. Bladder complications are very frequent, and various other visceral conditions; and from these, or bedsores, death results, not from the disease: perhaps the most frequent cause of death is pneumonia. Bulbar paralysis may supervene; but one of the most frequent of the nerve complications of tabes in my experience is general paralysis of the insane (paralytic dementia); and in visiting an asylum it is remarkable to learn what a large number of general paralytics have commenced as cases of tabes. Many varieties of transitional forms are met with in these two diseases among the out-patients, and give rise to much difficulty unless you remember the association.

With these preliminary remarks on the clinical history of this common disease we will now pass to the consideration of some examples of the disease. The first patient, J. W. M—, æt. 31, is a telegraphist. He went to my colleague, Mr. Dodd, for failure of vision in January, 1897, which proved to be due to grey atrophy of the optic discs. He also complained of diplopia, and his pupils presented the Argyll-Robertson phenomenon; the knee-jerk was absent, and upon inquiry it was found that three years before he had had so much difficulty in starting micturition that he was thought to have stricture. At the present time the bladder symptoms have disappeared, but the others have persisted, and he has lately had severe lightning pains, and he has complained of anæsthesia of the feet. There is no doubt whatever that it is a case of tabes, but you will observe that even yet there is not a vestige of ataxy in his walk. As regards the ætiology, he had syphilis twelve years ago, but although he has taken large doses of iodide for a considerable time they have apparently (as usual) done him no good. It is worth while noting that for three years he worked with a strong electric light two feet only from his head, and you will

observe that the earliest of his pronounced symptoms are connected with the optic disc. This is a very good illustration of the fact that prolonged forced functioning of any nerve structure tends to result in atrophy of that structure, a principle which we shall meet with again in the course of these studies. This patient, you will observe, has no ataxy, and although I cannot explain the fact I have frequently noticed that cases of tabes which start with lesion of the optic disc, either do not develop ataxic symptoms at all, or not until quite late in the history of the disease. But in spite of all our efforts he is now almost blind, though lately I believe he has somewhat improved under galvanism.

The next patient, J. G—, æt. 47, is one who also exhibits only pre-ataxic symptoms. He also applied to my colleague, Mr. Dodd, at the Westminster Ophthalmic Hospital for failure of vision due to an error of refraction, and, finding the pupils faulty in their action, Mr. Dodd suspected tabes and found his knee-jerks absent. The patient has the Argyll-Robertson pupil, and complains of a girdle of analgesia round his chest. These are the only symptoms which he has at the present time; but they are quite sufficient for a diagnosis, which is, however, confirmed by the history. There is no vestige of ataxy in his walk. This patient is a man of considerable intelligence, and there is a very clear history of his having had very definite ataxic symptoms ten years ago, from which he has entirely recovered. That illness commenced in 1885 with a difficulty of passing water, which was stated by his doctor to be due to a stricture, though the patient affirms that no stricture was found. He also had girdle pains, and severe lightning pains in the legs; there was a total absence of knee-jerk and loss of sensation in the feet, and at that time so much unsteadiness in the gait that he could not walk for more than ten or fifteen minutes at a time, and for some months could not use his hands to write. He subsequently went to Bath for treatment, and in view of the fact that he had contracted syphilis in 1880 he was treated for some two months with iodide. He did not take it longer than this, but continued the baths and other remedies, and completely recovered in the course of two years, remaining free from symptoms, as far as he is aware, for the past ten years.

Ætiologically this case is of the greatest interest. I have already referred to the fact that *prolonged forced functioning of any nerve structure results in its atrophy; it also results in the predominance and overpowering of it by the surrounding tissues, and a final degeneration of the fatigued structure.* This is sometimes known as Edinger's theory, but it is a principle with which neurologists have long been familiar, and is mentioned in the writings of our great compatriot Hughlings Jackson, as well as others. It is also met with in Cohnheim's statement, which I read to you, made nearly forty years ago. It is seen typically in the atrophic paralysis that accompanies various "occupation neuroses;" and it is found in operation in other diseases. It is a principle of the highest importance in neuro-pathology, and explains quite adequately, to my mind, the predominance of tabes in the male sex, who use their muscle-sense neurons so much more than females; and it also explains the fact that tabes comes on during the prime of manhood, at a time when the muscle-sense neurons are possibly exercised to excess. This patient illustrates this in a marked degree, for in the course of his occupation, that of a "directory canvasser," he walked immense distances, and was, to use his own phrase, "perpetually running up and down stairs." Added to this, two years before his first illness he got married, and indulged undoubtedly in much connubial excess. But, moreover, he had an alcoholic father, and we find in him an illustration of one of Charcot's favourite dicta, which runs as follows:—"Chaque goutte de liqueur séminale d'un alcoolique contient, en germe, la famille neuropathique tout entière." *

This, then, is the way we may state the different ætiological factors in his case. As a result of a predisposition, both inherited (alcoholic father) and acquired (syphilis), the nutrition of his nervous system was lowered and its power of resistance lessened; and by the exciting causes of his occupation (*i. e.* by forced functioning), and his sexual excesses, the nutrition of his muscle sense and other sensory neurons became specially impaired. I only saw him for the first time a week or ten days ago, but he is already improving under iodide and other remedies, a somewhat unusual circum-

stance, for cases of tabes of syphilitic origin rarely yield to antisyphilitic remedies—an unexplained but admitted fact.

The next patient, A. T. L.—, æt. 41, has been ill a much shorter time, but nevertheless he exhibits the symptoms of the ataxic stage. His illness began somewhat suddenly ten months ago with numbness of the feet and an unsteadiness in the walk. During the past year or eighteen months he has had transient double vision from time to time. At the present moment there is, as you see, well-marked ataxy in his gait, and Romberg's sign is also present. The knee-jerks are absent, and he has Argyll-Robertson pupils, but there is no alteration in the sensation anywhere, excepting a deficiency of the thermal sense of the right foot. He had syphilis seven years ago, and was at first put on iodide of potassium in large doses (small ones are certainly no use in these cases), but without any effect. More recently another method of treatment, which I shall mention to you directly, has been instituted, and he has already made some improvement.

The first and third cases, then, have improved slightly. The second recovered and remained well for ten years. These cases are in no way selected, beyond the fact that out of about ten or a dozen cases at present attending among my out-patients, they illustrate well the preataxic stage. There is a fourth case which I want you to study from the point of view of treatment, for in spite of drawbacks he has made a great stride towards cure.

F. A. W.—, æt. 35, came to me in October, 1897, complaining of "weakness" in his legs. It was in March, 1895, that he first felt a constriction round his waist, and nothing else appeared until January, 1896, when he had frequent attacks of gastric pain and vomiting. Ever since then he has complained from time to time that his feet felt "as though he was treading on nothing." Finally—it was not until March, 1897—somewhat suddenly, after one of the gastric crises, he manifested unsteadiness in the walk. You will remember having seen him in October last (1897), when the ataxy and loss of equilibrium were extreme, and the sciatic pains very severe. The knee-jerk was completely absent, and the Argyll-Robertson pupil was in evidence; the tactile sensation also at that time was delayed for as much as three or four seconds

* 'Nouvelle Iconographie de la Salpêtrière,' vol. v, p. 123, and elsewhere.

in the legs. I am glad you had an opportunity of seeing him then, for in the course of these three months he has amazingly improved. At that time he could not walk more than two or three hundred yards at a time, and now he can walk for over two miles with ease, and you can see for yourselves that the ataxy is much less. He can now feel the ground, and all his lightning pains have gone.

This brings me to the important question of *treatment*. Locomotor ataxy is often regarded as an incurable affection; and certainly many patients suffering from this relatively common and chronic malady go the round of London consulting rooms or hospitals, each time counting as a fresh case to swell the list of incurable nerve disorders. Undoubtedly the disease is difficult to cope with if not seen until an advanced stage is reached; but this is by no means the case if recognised early. The treatment adopted in the case I have just shown you is that which I have generally found the most successful, though it necessarily varies in different cases. The leading principles in his case have been the cessation of function of the deranged structures, a fairly liberal dietary, galvanism, and tonics in the shape of extract of malt and cod-liver oil. He has also been "suspended" a few times, and although there is no syphilitic taint, he took, for two or three weeks, five grains of iodide thrice daily. However, I doubt if the improvement could be traced to these, because they were not continuous, but they may have assisted. You will perhaps pardon my again insisting upon the importance of early recognition. If fortune favours us in this respect, I believe that of all the different methods rest of function occupies the front rank. If what I said about the theory of prolonged force functioning being so much in operation in this disease be correct, the *great rôle* which lies in store for this method of treatment as a restorer of damaged nerve structures or functions will readily be understood. This, however, is not the whole; the diet should be liberal, as just mentioned, and be specially directed to the nutrition of the nervous system; and it should be combined with massage or electricity, or both, according to the stage and variety of the case. Galvanism frequently applied was a favourite remedy with Erb, and he published many cases which improved more or less under its use. Applied with judgment in certain cases it is

certainly a most valuable aid, and I have one case in private practice that has unfortunately reached an advanced stage, which did not yield to other treatment, which is certainly improving under electricity. It is, however, a remedy capable of doing harm instead of good in unsuitable, *i. e.* "irritable" cases. Counter-irritation of the spine in advanced cases may be useful; but you will bear in mind that it prevents the application of electricity for some months afterwards. It frequently happens that we can only learn by trial which of the different kinds of treatment is most suitable. Belladonna and ergot may be given in cases where congestion of the cord is suspected from the presence of spinal or rapidly developing symptoms. Arsenic, silver, and other metals I believe to be positively injurious (with the exception, perhaps, of mercury hypodermically in some cases of syphilitic origin),—at least I have generally noticed that patients have gone back under their use. Didymine and cerebrin are two remedies which are still on their trial in such cases. So much for the curative treatment. There are certain things which should be avoided, namely, fatigue of mind and body, sexual excess, exposure to cold; and alcohol and tobacco should also be abandoned. Sea voyages are recommended by Sir William Gowers, but the patient should always avoid the falls to which travellers by sea are so liable, especially if unsteady on their legs.

The *symptomatic treatment* rests on general principles. The most important symptoms calling for treatment are the lightning pains and various other crises. In this connection the most important question is, shall we give morphia? and my answer is, unhesitatingly, no. Make it your study, gentlemen, to treat these pains by heat or by cold, by phenacetin or the other tar products, by galvanism or by massage, and by the many other means which modern therapeutics has placed at our disposal; but at all hazards save your patient from a life of thralldom, for with a recurrent pain like this the habit is bound to be developed. Moreover the same lines of treatment which cure or modify the disease also relieve the lightning pains; this was so in the last case I showed you. When you first saw him he was suffering very acutely, and was inclined to doubt the ability of a doctor who could not relieve them. But I persuaded him to have patience, and you

are witnesses of the happy issue. Therefore let us rather employ some temporary sedative, such as phenacetin, antipyrin, hyoscyamus, belladonna, physostigmin, or indian hemp, combined with rest, and perhaps warm baths (which often relieve pain), rather than launch the patient into a habit which gradually but surely leads to degeneration both of body and of mind.

The *prognosis* of tabes has hitherto been regarded as hopeless, at any rate for cure, though perhaps the disease may last throughout a patient's lifetime without materially shortening it. But as by degrees, by careful attention to clinical detail, we have learned to recognise it in its earlier stages, it is to be hoped, in view of the valuable researches to which I have alluded, that we may be able to arrest this degenerative process before it has spread from the arborisations to the neuraxon, and from the neuraxon to the neuron-body. The prognosis rests chiefly on three points:—first, upon the rapidity of development of the symptoms; secondly, on the causes in operation, and especially whether they be removable; and thirdly (the most important point), the stage at which the disease is recognised.

II. There are FOUR OTHER DISEASES of the spinal cord, all of which, compared with locomotor ataxy, are rare, which give rise to inco-ordination, and therefore a disorder of the gait resembling tabes. We must briefly consider the means of differentiating them from it before we pass to the consideration of cerebellar tumour. They are spinal tumour (lumbar region), spinal pachymeningitis, Friedreich's disease, and ataxic paraplegia.

1. *Spinal tumour*.—Tumour of the spine in the lumbar region is apt to be attended by ataxic symptoms which so precisely resemble those of tabes that they may readily be mistaken. In most cases, however, this disease may be distinguished from tabes, *first*, by the absence of the pre-ataxic symptoms before mentioned; *secondly*, by the fact that it is preceded by severe, *constant* neuralgic pains in the spine, spreading upwards and downwards from the lumbar region; and *thirdly*, as in all cases of vertebral or spinal tumour, all the symptoms—which, be it noted, include actual weakness and often stiffness of the legs—have a tendency to be unilateral, *i. e.* to predominate in one or other leg.

2. *Spinal pachymeningitis*, or chronic thickening of the meninges, is apt sometimes to be confined to or at any rate to predominate in the posterior region of the theca vertebralis; and in that case gives rise to symptoms which so closely resemble tabes that it is quite possible, I believe, that many cases of so-called tabes cured by iodide were in reality cases of this affection of syphilitic origin. The condition I refer to is extremely chronic, and not infrequently the symptoms are few and may be ill-marked, on which account such cases very rarely find their way into hospitals. They get into the infirmaries, however, and my experience at the Paddington Infirmary led me to the conclusion that the disease is a fairly common one. At a meeting of the Neurological Society held at the Paddington Infirmary in 1890 I was able to show the cords of five fatal cases, and three cases believed to be examples of the affection during life.

The spinal cord which I show you was taken from a woman who was admitted into that infirmary in January, 1891, with all the classical symptoms of tabes, preceded and accompanied by very severe "lightning pains," excepting that the patient was a woman, and that she was seventy-one years of age. The history, briefly, was that for three years prior to admission she had suffered from paroxysmal pains shooting down the legs and various other places, and that she had had other attacks which resembled gastric crises. For two years prior to admission she had suffered from ataxy, which became progressively worse, and at last so marked that she was unable to stand. Many careful observers saw the case; all had no doubt that it was an example of locomotor ataxy, albeit in a woman. She died of pneumonia in June, 1891, and I will quote a brief passage from the report on the autopsy. "Spinal column: the parietal theca vertebralis seems to be normal, and of normal thickness. Pia mater and arachnoid on the anterior surface of the cord also normal, but on the posterior surface is considerably thickened and opaque. This thickening is irregularly distributed from end to end, and is very much more marked in two patches of about three inches in length in the mid-dorsal and in the lumbar regions, in which position it is about the thickness of a piece of wash-leather. Throughout the entire length of the cord the thickened meninges are lightly adherent to the dura, and firmly adherent

to the cord beneath along the posterior columns, especially in the lower half." Under the microscope you will see a section of the cord. There was sclerosis of the posterior white columns, in the position corresponding to the thickenings of the theca which were adherent to the diseased column of the cord; and the symptoms were doubtless due to the spread, by continuity, of the inflammation from the meninges to the posterior columns.

I know of no means of recognising this affection with certainty from tabes, but one is assisted if there is a long history of *continuous* pain and tenderness in the spinal column, and *continuous* pain stretching down the spinal nerves; and sometimes by the age and sex of the patient.

3. *Friedreich's disease* need only be mentioned here.

4. *Ataxic paraplegia* is another rare affection, which corresponds in my belief to the spasmodic tabes dorsalis of some authors. It is due to sclerosis affecting the posterior and the lateral columns, and you get the *symptoms of these two lesions*. It is differentiated from tabes by the rigidity, increased knee-jerks, and ankle-clonus. Here is a typical case which is now almost well, for the disease had a syphilitic origin, and the patient has been under treatment with iodide for over six months.

III. CEREBELLAR TUMOUR is often very obscure, but it may be suspected in presence of a group of four symptoms, namely, a reeling gait, headache, vertigo, and nystagmus, especially when these symptoms occur in childhood. The characteristics of the walk I have already mentioned to you. It is that of a drunken person who traces a sinuous line along the floor as he walks along. There is frequently weakness, but rarely actual paralysis; sometimes hemiplegic, but more frequently paraplegic or general in distribution. The knee-jerk does not help us very much. It may be absent, or increased, or normal. The cranial nerves most frequently affected are the sixth and the optic nerve. Symptoms occasionally present are,—forced movements of the neck, for example, pulling down the head towards the side on which the lesion is situated, in tumour of the lateral lobe; and other occasional symptoms I have seen are attacks of syncope, or of palpitation and dyspnoea, which are of considerable gravity, as

they usually indicate pressure on or spread to the medulla, though in one instance of this kind the patient (on whom I made the autopsy) lived for ten or twelve months.

The patient who is coming in is a woman named D—, 26 years of age. She has been married eight years, and was quite well until three years ago, when her walk gradually became unsteady—the legs, she says, became weak; and you will see now that there is very curious inco-ordination of both the legs and the arms. The walk is peculiar. It resembles tabes somewhat, but is not typical. The legs have not the exaggerated action. Careful search in her case reveals no other symptom of any note. The pupils are apparently normal, there is no diplopia. I am not quite sure about nystagmus, and the optic discs have not yet been examined, for she only came to the hospital yesterday for the first time. She has had headache from time to time, and there is some doubt about vertigo and nausea. The case certainly resembles tabes in some respects, but against it are the age and sex, the absence of any of the pre-ataxic symptoms, the involvement of the arms, and the walk, which is not typically ataxic, although the disease, whatever it is, is considerably advanced. And if you notice carefully, you will see that she has at times a tremor of the limbs when they are put in movement. The diagnosis is not sure, but in my belief it is very probably cerebellar tumour.

IV. The walk is also more or less characteristic in the following diseases, of some of which I am happy to be able to show you examples.

(1) Here is a case of *paralysis agitans*. In this the patient is continually stooping forward, and shuffles along the floor with his enfeebled and rigid limbs, his face looking fixedly forward all the while.

(2) Here is a case of *spastic paraplegia* dating from birth, though the patient has been much worse lately. He is now forty-three, and you will observe that the limbs are stiff, and he has a tendency to the "cross-legged progression" which is characteristic of advanced cases of this disease. In cases of *lateral sclerosis* of cerebral origin the same characteristics may be observed, only then they are confined to one side. In certain cases of spastic paraplegia of long standing like one I saw to-day there is a tremulousness of the legs which

is very peculiar. The walk in such cases can only be described as "wobbly," if you will excuse the expression. It presents a very marked contrast to the "rigid" walk, which is more usual.

(3) In *hysterical hemiplegia* Brodie long ago pointed out that the affected limb trails along the ground behind the patient. This feature formed in his belief a valuable means of differentiating cases of functional from organic hemiplegia, and certainly in organic cases attended by descending lesions the leg is stiff, and is circumducted as the patient walks along. But it must be remembered that this distinction only applies to hysterical hemiplegia in which the paralysis is flaccid. Some cases are attended by rigidity, and then the distinction does not obtain.

(4) In *alcoholic paralysis* (peripheral neuritis) there is foot-drop, and this gives rise to a kind of "high-stepping" action which is very typical. In marked or advanced cases, the falling of the toes when the patient by an effort raises the foot is extreme, and he may find it impossible to go upstairs.

(5) In the *diphtheritic paralysis* of children (another form of peripheral neuritis) the walk, or rather the attitude of the patient, is so characteristic that one can often detect the disease as he enters the room. He hangs his head forward from weakness of the neck muscles, and the "flabbiness" of all his movements is peculiar. I am not aware that attention has been called to this fact, but it has often rendered me signal service where the history is obscure; and on inquiry you may find that fluids return through the nose, and there have been cases of "sore throat" in the house.

(6) The typical walk, or rather dance, of *chorea* you have seen on former occasions. Many of the other diseases attended by tremor which we were considering in a previous lecture, give rise to an uncertainty of movement of the limbs which renders the walk peculiar.

(7) Partial weakness from old age, *general debility*, or other cause gives rise to the stumbling gait with which we must all of us be familiar.

(8) The characteristic loss of equilibrium in the *vertigo* of some aural, ocular, and circulatory affections is only met with during the attacks. These will be considered on a future occasion. Time does not permit of our pursuing this interesting topic further.

In conclusion, gentlemen, if you will think over

our studies of to-day, and the cases we have seen together, I believe you will agree with me that we have in *tabes dorsalis* a malady, chronic, it is true, but often curable, or at any rate amenable to treatment under appropriate measures, especially if recognised early. We have, therefore, one more malady which must be taken out of that hopeless class which some maintain—in my humble opinion incorrectly—forms the bulk of nervous disorders.*

* Since this lecture was delivered I have received several sceptical letters from medical men, the latest of which says, "I have never seen locomotor ataxy get better, and I fail to see (*prima facie*) how such a degenerative change can improve." My reply is an appeal to facts. Up to date (April 22nd) I have treated eleven cases on the methods (in various proportions, for no two cases are just alike) advocated above. Here is the latest case, still under treatment. Mr. —, æt. 51, was brought to me by Dr. Maitland Thompson: had girdle pain as long ago as 1884; ataxy came on four years ago; unable to walk without sticks, and then only the length of his garden; pupils very unequal and immobile to light and accommodation; loss of knee-jerks; anæsthesia, analgesia, and allocheiria up to knee both sides; hyperæsthesia round waist; frequent lightning pains; occasional "crises gastriques," &c. Has only been under treatment 3½ weeks: hyperæsthesia has gone; sensation has returned everywhere except part of sole, right foot; lightning pains only once; no crises gastrique; evidence of returning muscle sense in legs and feet; walk improving; *pupils more equal, both react slowly but evidently to accommodation, right one reacts slightly to light*; general health greatly improved.

Peptonuria in some of the Acute Infectious Diseases of Children.—C. Cattaneo ('Jahrb. f. Kinderheilk.) examined for peptone the urine of twenty-five children, of whom six were suffering from measles, five from scarlet fever, thirteen from diphtheria, and one from erysipelas. In no case was there any condition such as suppuration, intestinal disease, &c., present which might cause peptonuria independent of the infectious disease. In all but two cases peptone was present in the urine on the first day, gradually disappearing (present on second day in ten, on third in seven, &c.). In one of the two cases in which it was absent on the first day it appeared on the second. The author explains the regular appearance of peptonuria on the first day by the fact that the children each received an anti-diphtheritic serum inoculation on admission. As a result of 123 examinations made in the twenty-five cases, twenty-nine being positive, no practical conclusion can be drawn. The appearance of peptone was irregular, and in no way related to the severity of the case. In one child only did peptonuria occur constantly; this was a mild case of measles in a child with very severe rickets. As far as the author knows no investigation in this line has been made in rachitis, but he thinks it offers a very promising field of work.

Pediatrics, April, 1898.

THE SURGICAL AFFECTIONS OF THE STOMACH, AND THEIR TREATMENT.

A Course of Lectures delivered at University College during February, 1898, by

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No. III.

WE now turn to the surgery of the non-malignant diseases of the stomach, a branch of our subject which is beginning to receive very careful attention in this country, and one which is far more important and interesting than those which have gone before. Whatever may be said of the attitude of English surgeons towards the question of the treatment of malignant disease of the stomach by operative procedures, there can be no doubt as to their activity in dealing with perforating ulcer. Our attitude in regard to the surgical treatment of the conditions depending upon ulcers, and simple pyloric stenosis with dilatation and adhesions and gastralgia, is still one of great caution; we are slow to adopt the reasoning which is put forward abroad in regard to these cases, and much slower to undertake the operative treatment of gastric disease as a *preventive* measure. But about perforating ulcers we do not hesitate. Quite a large number of such cases have been operated on in the United Kingdom, and with results which are most encouraging. About a year ago I commenced collecting these native cases for a special purpose. But when Mr. Goffe, not long ago, asked me to give him some task of the kind, I gratefully handed over the completion of the list to him, and he has spared no pains to make it as full as possible, and to bring it down to the present date. I do not intend to ask your consideration of the injuries of the stomach, such as stabs and bullet wounds. The surgical treatment of these is now on a sound footing, and needs no special description. It consists in antiseptic abdominal section through the existing wound as soon as possible after the injury, the cleansing of the abdominal cavity, and the closing of the aperture in the stomach, with or

without paring of its edges. The question of drainage by tube or gauze must be settled in each case separately on its own merits, but in most cases when operated upon early no drainage of any kind is required.

A consideration of the surgery of the non-malignant diseases of the stomach divides itself naturally, according to the most modern ideas, into three parts (Table XI):

TABLE XI.

Operations for Non-malignant Disease of the Stomach.

Group I.—Emergency Operations:

- (a) Gastrorrhaphy for Perforation.
- (b) Gastrotomy for Hæmorrhage.

Group II.—Remedial Operations:

- (a) Divulsion for Pyloric Stenosis.
- (b) Pyloroplasty for Pyloric Stenosis.
- (c) Gastroplasty for Hour-glass Stomach.
- (d) Gastro-anastomosis for Hour-glass Stomach.
- (e) Gastroplicatio for Dilated Stomach.
- (f) Gastrolysis for Adhesions.

Group III.—Preventive Operations:

- (a) Pyloroplasty for Pyloric Stenosis.
- (b) Gastro-enterostomy for all the above.

(1) Those measures which are undertaken for serious accidents occurring in the course of gastric ulceration, such as perforation or hæmorrhage, which may be called *emergency* operations. (2) Those measures which aim at remedying the more serious chronic evils due to the same, which might be called *remedial* operations. (3) Following the most recent views of all, there are a series of measures which aim at *stopping* the more chronic gastric disturbances from becoming dangerous to life or from disabling the patient, which measures may be called prophylactic or preventive operations. Under the first heading of emergencies will come gastrorrhaphy for perforation, gastrotomy for hæmorrhage, either alone or combined with gastro-enterostomy, which of course relieves the stomach very considerably in an early stage of digestion, and consequently keeps the ulcer for which the treatment has been adopted from the irritating effect of half-digested food. That way of treating mere chronic gastric ulceration or gastric inflammation is coming more and more into vogue,

and is yielding better and better results, as we shall see presently when we come to consider the preventive operations. But even in the earlier emergency operations gastro-enterostomy has its place, and I shall have to allude later on to a case operated on by Küster, in which he successfully treated two cases of gastric hæmorrhage by securing the artery through an incision in the stomach, and then doing gastro-enterostomy. Under the second heading, "remedial operations," we come to quite a series of procedures. First of all pyloric divulsion, through an opening in the stomach, which I have told you about before as having been designed originally by Loretta with a modification by Hahn, who succeeded in stretching the pylorus without incising the organ. This is done for pyloric stenosis. Then we have pyloroplasty (Fig. 7), or enlarging the outlet by longi-

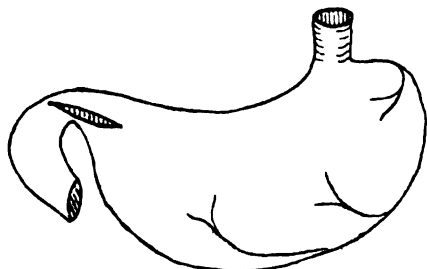


Fig. 7.—v. Heineke's pyloroplasty. 1st stage.

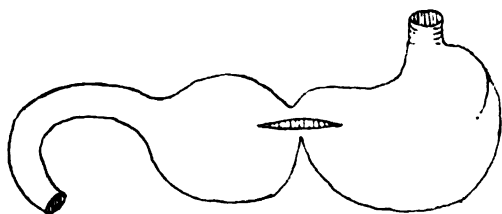


Fig. 9.—Gastroplasty. 1st stage.

tudinal incision in the cross-suture, also for pyloric stenosis. We have gastroplasty for hour-glass stomach (Fig. 9), which is a form of stenosis of the organ, and a very serious one. Another operation, also included in my list, is gastro-anastomosis, which I will briefly describe by the aid of this diagram, Fig. 11). You have a stomach contraction here, which causes dilatation of the proximal portion. You can do gastroplasty for this as I have explained, or you may do this gastro-anastomosis, first performed by Woelfler.

An opening is made in the two portions of the organ on either side of the constriction; these are

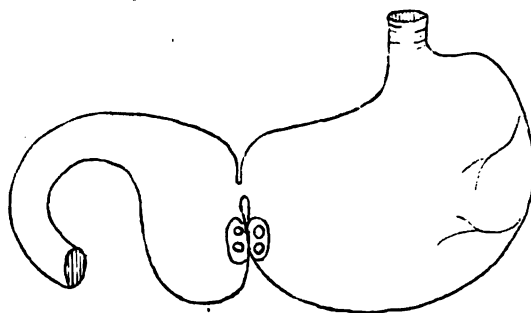


Fig. 11.—Woelfler's Gastro-anastomosis.

then united together, either by a Murphy's button or by ordinary suture.

There is also gastroplication, an operation which is likely to be cast aside as unscientific, namely, the folding up of the stomach and reducing its bulk. It is, however, simply a question of treating the symptom rather than the disease. Gastrolisis is a very important operation indeed, although perhaps it hardly deserves a special description. It consists in the removal of adhesions between the stomach and the adjacent parts by operation, these adhesions being, in the opinion of a great many surgeons and physicians, including our own professor of medicine, Dr. Roberts, the cause of very severe pains and other gastric disturbances.

Under the third heading of preventive operations (Figs. 5 and 6), now coming to the front, we have pyloroplasty and gastro-enterostomy, for the relief of morbid changes which are sure in a great many cases to lead to some of these conditions which we have been considering. There are for these both the anterior and posterior methods.

At the end of my last lecture I alluded to the fact, which I should like to repeat here—that whatever may have been the attitude of surgeons in this country towards operative procedures for the relief of malignant disease, in which we have been very slow, perhaps wisely, to adopt the very daring operations which have been performed elsewhere, there is no question whatever as to the attitude of English surgeons towards the life-saving operations for non-malignant disease. That fact will come out in our tables presently. But our attitude towards the remedial operations

has been one of very great caution, while in the direction of preventive operations we have practically done nothing, whether rightly or wrongly I do not say.

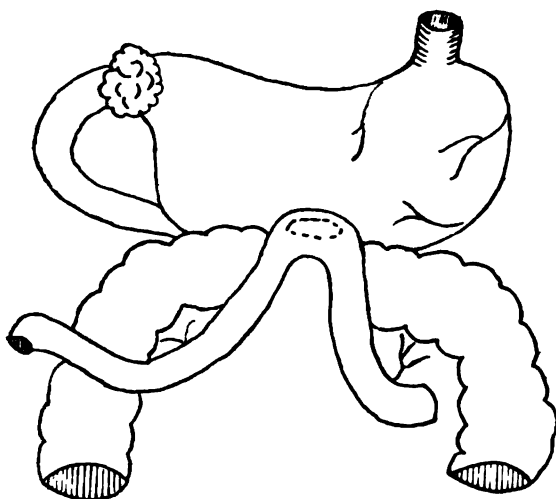


Fig. 5.—Woelfler's anterior gastroenterostomy.

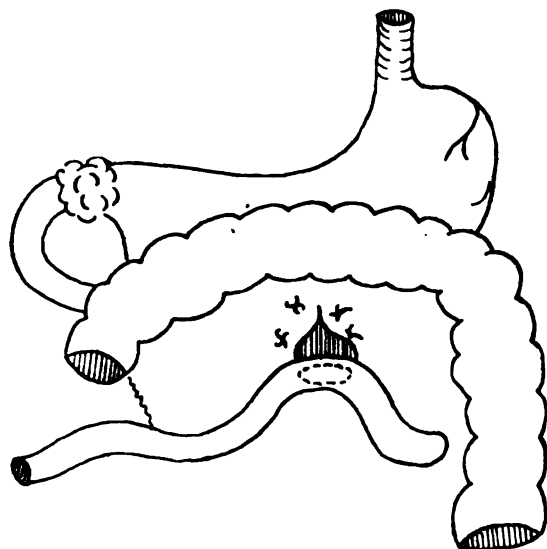


Fig. 6.—v. Hacker's posterior gastro-enterostomy.

I do not intend here to refer to the clinical history of gastric ulcer, nor do I intend to allude to its pathology; these matters belong rather to medicine than to surgery, and Dr. Roberts agrees with me that it is the right course to take. Dr. Sidney Martin, too, tells me he takes the more detailed pathology of the affection.

Now it is not quite certain who was the first

to successfully operate for perforated gastric ulcer, but the earliest case I can find recorded is attributed to Heusner, of Barmen. In this country the first success appears to have been achieved by Dr. Lundie, a northern surgeon, in 1893. In our own hospital the earliest success was in 1895, at the hands of Mr. Pollard.

In examining the results of operations for perforated gastric ulcer, I will first take all the cases operated on in England and America which Mr. Goffe has been able to collect in the list before me. The study of this brings out many interesting and important facts, on which many conclusions as to treatment can be based (see Table XII). I have also to show you a short analysis of a large number of these operations from all sources made by Mikulicz up to the end of last year, which includes very large lists by Pariser and Wier and Foote, and may be taken to include all the Continental cases to date.

In this Table XII you will see two or three interesting facts. Altogether 103 cases were collected, including American, English, and Continental. Mikulicz puts them under two headings, according to the time at which they had been done. From 1885 to 1893 there have been operated on 35 cases, of which 34 died and 1 recovered, giving the percentage of deaths of 97.15. From 1894 to 1896 there were 68 operations, and assuming that these included all which were unsuccessful—which is very doubtful—we find there recovered 32, and died 36, giving a mortality percentage of 52.94. Over the whole period 33 recovered and 70 died, giving a mortality of 67.96 per cent.

Table XII (from Mikulicz).

Statistics of hitherto published Cases of Perforated Gastric Ulcer.

Year.	Number.	Recov.	Died.	Mortality.
1885 to 1893	35	1	34	97.15
1894 „ 1896	68	32	36	52.94
	103	33	70	67.96

These figures include those of Pariser and Wier and Foote.

Now I come to Mr. Goffe's table, arranged for me in somewhat the same way, of perforated ulcer

treated by operation in England (Table XIII). You will observe that he has been able to collect exactly 110 cases. The deaths amongst the 8 males were 5, showing a mortality of 62·5 per cent. The deaths among 77 females were 34, a mortality of 44·1

Table XIII (Goffe).

Perforated Gastric Ulcer treated by Operation in England.

Sex.	Number.	Deaths.	Mortality.
Males	8	5	62·5 %
Females	77	34	44·1 %
(?)	22	16	72·7 %
	107	55	51·4

In 3 the result is not stated.

per cent. Then there were 22 cases in which the surgeons had not thought it necessary to say whether they were operating upon males or females, and of these 16 deaths, mortality 72 per cent. In 3 the result is not stated. It is a remarkable fact that there are still a great many people who are very slipshod in their records, taking it for granted that one follows their own line of thought, and that it is not worth while recording this or that fact about their case whether the patients are old or young, or whether they die or live. Another table, arranged by Mr. Goffe (Table XIV), includes operations done in Canada and the United States. In

Table XIV (Goffe).

Perforated Gastric Ulcer treated by Operation in Canada and United States.

Sex.	Number.	Deaths.	Mortality.
Males	2	2	100
Females	13	5	38·4
(?)	3	—	—
	18	7	38·8

that part of the world there seem to have been only published records of 18 cases, as far as could be made out, most of which (13) were females. It is a remarkable fact brought out by all these figures that there is an enormous preponderance of females among those suffering from gastric ulcer. In another table it will be apparent that perforations

in females occur under thirty years of age as a rule, while in males the subjects are mostly men over thirty. In the present table you see there are only 2 males operated on, both of whom died; 13 females were operated on, of whom 5 died, a mortality of 33·6 per cent. There were 3 whose sex was not recorded, and they all recovered. The average mortality for all of these was 38·8 per cent.—a very respectable state of things supposing that all the cases, whether successful or not, were recorded—which again I think is doubtful.

Now the first matter of scientific importance which arises out of these statistics is the great industry of Mr. Goffe, who has gone to a very great deal of trouble, and I think I may say that, like most of those who have gone into a thing with all their heart, he has enjoyed it very much, and has produced a record from English sources which is fuller than any I know of up to the present day. He has also brought out a very interesting fact, namely, that in England alone we have over 110 cases certainly to our credit, and that only last year all the cases collected by Professor Mikulicz from all the countries of the world only produced 103. That plainly shows that we have not been slow in practising this life-saving branch of surgery. It also comes out in these statistics that we have had a very fair measure of success. At all events, it is perfectly clear that 55 patients have been saved from inevitable death by prompt surgery, who a few years ago would have been certain to lose their lives; for of all the accidents which can befall the alimentary tract, perforation of the stomach by disease is perhaps the most fatal. Few or no cases recover from it without operation. Perhaps here and there, where adhesions take place in cases where the perforation has been very slow in formation, one or two patients have survived; but where clean-cut perforation has taken place through the stomach as the result of ulcer the patients are doomed to certain death, and that very speedily. And though the mortality, even where the surgeon is called in, is still very high, nevertheless the fact remains, as you will see from this and other tables, one in every three patients is saved, perhaps more. Out of those nine cases upon which I have operated, three made a perfect recovery, two more were very nearly saved, living respectively fifteen and ten days, and dying ultimately not of

the original condition, and not of the perforation, but of complications, to which you will see presently these patients are peculiarly liable. The next point which is attested by our list is that for success these cases must be early diagnosed, and *at once* placed in the surgeon's hands. And here of course we look to the physicians and to those who are in general practice to determine the fate of these patients in the future. They will fall into their hands in the first instance. If those gentlemen are clear in their diagnosis and prompt in their action, a great many of these unfortunates will be saved. If such be not the case the subjects of the condition must inevitably die. Three factors in the history of these cases appear to exercise a most important influence on the success of the operation which is undertaken for their cure. (1) The character of the last meal or meals. (2) The time after the last meal at which the perforation took place, and the immediate treatment of the patient by the practitioner who was first called to the case, or by the friends of the patient. (3) The time which has elapsed between the moment of the perforation and the beginning of the operation. In reference to the first of these points, it is clear that if perforation has taken place after a light digestible meal, say consisting of an egg and bread and butter and weak tea, or milk, there will be less material to escape in the first place from the stomach into the peritoneum. And in the next place, what is extravasated will be comparatively unirritating and probably but slightly if at all septic. This was found in one of my own cases which recovered. She had recently had a very light meal—an egg and tea and bread and butter, and gone to bed a couple of hours afterwards; perforation took place while she was going to sleep. Therefore she was under the most favourable conditions for recovery. If she had been in the vertical position the fluid would have been extravasated all over the abdomen. If the meal is a large one and contains indigestible matters, not only is there more material to escape from the perforation, but the stomach will make a great effort to expel it, and much will be forced out into the peritoneum. What escapes will probably be essentially septic and more or less decomposed, and the amount of hydrochloric acid will be too small to render the substances in the stomach antiseptic, but yet sufficiently strong to irritate the

peritoneum. You will remember that on an indigestible meal the stomach contracts firmly, and some of us know occasionally the sensation of indigestion, which is in a great measure due to the fact that the stomach is trying to force on indigestible matter against the pylorus, which stands guard on the stomach to prevent matter passing into the duodenum before it is duly reduced to fluid or pulp. In the abdominal cavity of one of my patients I found a considerable part of a recent meal, consisting of bacon, greens, and potatoes, and there were fragments of these amongst the intestines. The effect in this case was most violent peritonitis, from which the patient died rapidly, as was to be expected.

Now in reference to the second point—the time after the last meal at which the perforation took place. It must be evident that a patient has a better chance of being in a good condition for operation if the meal has been digested, and has passed into the small intestine, than if it has been quite a recent meal. In the first place, such patients are stronger to bear the severe strain of the operative measure and the subsequent starvation, which has to be most strictly carried out afterwards, and there will probably be less food in the stomach to escape into the peritoneum if a considerable time has elapsed since a meal was taken. This is also well illustrated in the lists of recoveries.

But perhaps the factor which more than all determines the success or failure of these cases is the time which has elapsed between the moment of perforation and the operation. This comes out

Table XV (Goffe).

*Time lost from Perforation to Operation.
English Cases.*

Hours.	Cases.	Deaths.	Mortality.
Up to 12	42	10	24·0
12 to 24	16	12	75·0
Over 24	27	19	70·4
Not stated	28	—	—

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clearly from our statistics. From Mikulicz's figures it appears that the chances of recovery are four times as great when the operation is done within the first twelve hours as when it is done later.

The same is seen from Mr. Goffe's tables from English and American cases, which I will show you now (Table XV).

The time lost from perforation to operation in English cases has been arranged in periods of twelve hours. But there are 28 cases in which the time is not stated. Time lost up to twelve hours shows 42 cases with 10 deaths, mortality only 24·0 per cent. Twelve to twenty-four hours 16 cases, 12 deaths, mortality 75 per cent. Over twenty-four hours 27 cases, 19 deaths, mortality 70·4 per cent. There can be no doubt that out of these 28 cases in which the time is not stated, there would have been so many deaths as to considerably raise the mortality of the last group. They probably all died. Next we have the American cases (Table XVI), which are not so

Table XVI (Goffe).

*Time lost from Perforation to Operation.
American Cases.*

Hours.	Cases.	Deaths.	Mortality.
Up to 12	2	—	—
12 to 24	5	2	40
Over 24	7	5	71·4
Not stated	4	4	100
	18		

numerous; there are 4 in which the time is not stated, and all of them died. Those who were operated on less than twelve hours after the perforation recovered, namely, 2. From twelve to twenty-four hours there were 5 cases, of whom 3 recovered, mortality 40 per cent. Seven were operated on after twenty-four hours, and 5 of these died, mortality 71·4 per cent. The Continental figures, which are not so large, show exactly the same thing. I have also put here my own cases (Table XVII), which you will forgive me for saying is the only series of more than twos and threes done by a single operator in one institution, which have a value of course, because they are not merely selected cases. We have 9 cases, 8 of whom were operated on in the hospital, and one elsewhere. There were 2 males, 7 females. Both the males died. Of the 7 females 4 died, and the aggregate mortality was 66·6 per cent., which is far more likely to be an accurate estimate of the fatality

which occurs than if the records were collected from a very much larger number of cases picked up from the journals (Table XIV). The time lost in these cases of mine is seen below. Up

Table XVII (myself).

Perforated Gastric Ulcer treated.

Sex.	Cases.	Deaths.	Mortality.
Males	2	2	100
Females	7	4	57·1
	9	6	66·6

to twelve hours 5 cases were operated on, and 2 of these recovered, the mortality being 40 per cent. One case was operated on between twelve and twenty-four hours—as a matter of fact eighteen hours,—and that case recovered. Three cases were operated on after twenty-four hours; they all died, which brings the mortality up to 66·6 per cent. It is only when we have a long series of cases done by a single surgeon in one institution, under the same conditions, and supplied by the same class of practitioners, and by the same colleagues who recognise the need of early operation, that we shall really be in a position to say what the mortality of these cases really is. Mr. Goffe tells me, in going over these figures, that there is no other table showing so many cases from the practice of one surgeon anywhere, and that shows how rare the cases are. That this loss of time should be serious is only what one would expect.

Table XVIII (myself).

Time lost from Perforation to Operation.

Hours.	Cases.	Deaths.	Mortality.
Up to 12	5	2	40·0
12 to 24	1	—	00·0
Over 24	3	3	100·0
	9		

I have no doubt you have been told, when considering the clinical symptoms of gastric ulcer, that one of the most striking is the abolition of the liver dulness by the escape of gas into the peritoneum. Doubtless that is so, but not only does the gas escape from the stomach into the abdomen in

these cases, but it is probably developed in the abdomen besides by decomposition, and the consequence is an amount of tension on the abdominal walls which is most startling. The effect of this tension is to force up the diaphragm and so press upon the pericardium and the lungs, and cause very serious embarrassment of cardiac action and to respiration. These patients suffer very much from this condition of things; they have very rapid pulses and laboured breathing, and very often an irregular heart. The inhibitory effect of distension upon the heart is a point which has to be reckoned with, and if the operation is done early this is to a large extent avoided. Moreover the operation will not be so severe where only a short time has elapsed since the perforation, for the contents which have escaped will not be spread very far, and the area of cleansing will be limited to the anterior part of the abdomen, so that both time and the patient's strength are saved. Where a long time has elapsed the contents of the stomach are almost sure to be widely distributed through the abdominal cavity, and to have found their way into the pelvis and into the flanks above the liver on both sides, round the spleen, where it is very difficult to dislodge, and so on. That means that we have to adopt the most elaborate measures, either of flushing or sponging, in order to cleanse the peritoneal cavity. The demand made upon the patient's strength in these cases is simply enormous. In one of my own cases the extravasated matter had found its way into both flanks, above the liver on both sides and into the pelvis, from which it was removed by a sponge with the hand thrust into all these situations. The operation consequently lasted fully two hours. To my amazement the patient recovered. The time taken in my other cases was as follows, and I give you this because I know the statements are accurate, and because the time that the operation lasts is not given in many records.

I occupied 2 hours, and recovered.

I " 1 hour 25 minutes, and recovered.

I " 1 " 23 " died.

I " 1 " 15 " "

I " 1 " 5 " "

I " 1 " 15 " "

I " 1 " 20 " "

I " 1 " and died.

Here, of course, you will notice something that

appears to rather conflict with what I have stated, namely, that the cases which recovered in this list took the longest time to operate upon. This is probably due to the fact that in the other six cases four were obviously quite hopelessly feeble when I began, and I hurried my operation, feeling that if I did not do so the patient could not possibly recover. One, my first case, died subsequently of a subphrenic abscess on the fifteenth day, due to the fact that I had not thoroughly cleansed the subphrenic space with my hand in the first instance. Another died on the tenth day, when he had perfectly got over the operation, and had not had a bad symptom. He died because the original ulcer perforated an artery, and he bled to death into the stomach.

Now before considering the operation itself I ought to point out to you that in these cases we ought not to wait for the shock to have passed off. Many of these patients when we first see them are suffering from collapse, and the great nerve centres in the abdomen are tremendously influenced in the direction of inhibition, owing to the perforation and the extravasation among the viscera. It is a question whether in such cases the surgeon ought to interfere or not. There are those who hold that he ought not, but surgeons have lately come round to the view that he ought. As a rule you are obliged to wait a short time, until you have prepared everything for the operation, and that is about as long as we ought to delay. In some cases, however, there is no shock; in one or two of my cases this was so. Where shock is present it is due in a great measure to distension by gas in the peritoneum pressing upon the diaphragm and embarrassing the heart's action and the lung; it is also due to the irritation of the peritoneum by substances extravasated into it. Now both these causes are removed by opening the abdomen. You cut down as quickly as you can, and when you see the peritoneum, before it has been opened, it generally bulges into the wound, and you notice that it is distended with gas. If you are wise you will make a prick in it to allow the gas to escape slowly. If the opening be not larger than a goose-quill the gas will rush out with a force which will clearly show the amount of tension. In one case I held my hand six inches from the hole, and felt the gas whistling against it for about half a minute before the abdomen was empty. As soon as the

gas has escaped the fluid begins to run out, and in some of my operations I have been struck by the marked improvement in the pulse, respiration, and the general condition which was noted by the anæsthetist when the gas and irritating fluid were allowed to escape through the incision. After all, the division of the abdominal wall is a small matter. It ought not to add to the shock, and if patients are capable of bearing an anæsthetic at all they are able to bear this incision in addition without any prospect of an increase of collapse. Of course the subsequent handling of the intestine is likely to produce very extensive shock or to increase that already existent, and that we cannot avoid. For this reason we should avoid all unnecessary pulling on the stomach subsequently.

This leads me to the question of the operations done for gastric ulcer. Fortunately for us, most of these perforations take place on the anterior surface of the stomach. An analysis of large numbers of the cases which we have here will show that in 77·1 of them the ulcers were on the anterior surface, the other 20 per cent. being on the posterior surface. Practically the same proportions are seen in our table of English and American cases (Table XIX).

Table XIX.

Site of Perforation of Gastric Ulcer treated by Operation—English and American sources.

Site.	Cases.	Percentage.
Anterior wall	67	77·00
Post. wall.....	11	12·64
Lesser curve	9	10·34
Total	87	

In 27 cases the site was not recorded, and in 4 cases no perforation was found.

As to the operation itself: the patient is prepared, as for all extensive abdominal sections, in such a way as to ensure him against chill, namely, by wrapping him up in wool, putting hot bottles about him, or placing him on a heated table, for he has to face a procedure about as exhausting as any operation could be. Of course it goes without saying that we cleanse the skin thoroughly. Every other preparation is made beforehand for the saving of time, even to the

threading of your needles, &c. You must get your assistant to make it his aim to see that everything is quite ready. The place for the incision in the abdominal wall is the first matter. It should be in the mid-line above the umbilicus in almost every case. And let me advise you to follow the rule which is adopted, I think, by most surgeons in these cases, namely, to make your incision in the first place comparatively small,—that is a fairly liberal skin incision, a less extensive division of the muscular wall, and a very limited peritoneal incision, just enough to enable you to get in your finger. Always remember that we may make an error in diagnosis—there may be no perforation; such things as that have happened, and it is very desirable that we should not have opened the abdomen widely to find that we have made a mistake. When we have opened the peritoneum, if we clearly see the fluid and hear the gas bubbling out we are sure of our position. When the gas and fluid have nearly rushed out, we take our scissors and make the opening as large as we want to. We see the stomach coated with lymph as a rule, and usually the direction from which the fluid is coming. If the incision which you have made is large enough to get access to the ulcer, well and good; if not, you must divide the rectus on one side or another: usually it is the left rectus which is divided transversely to the first cut. That will give you free access to the part.

(To be continued.)

Hernias during Pregnancy and Parturition.

—H. Fischer has been studying the twenty-four cases on record with eight of his own, and confirms the general assumption that severe hernias under such circumstances are particularly troublesome, although the usual rest, &c., of the women favours the cure of slight pre-existing hernias. He has seen two cases of femoral hernia entirely cured with an English truss and the reclining position during pregnancy, and also cases of umbilical hernia held with strips of plaster, over which a good fitting bandage was worn. He does not believe in postponing a necessary operation on account of the pregnancy or childbed, but advises an immediate operation after delivery, without waiting for the collapse from incarceration to supplement the operation shock, always using the Schleich method on account of the danger from vomiting after general narcosis, which produced abortion and death in one of his pregnant cases.—*Deutsche med. Woch.*, March 3rd; *Journ. Amer. Med. Assoc.*, April 9th, 1898.

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A CLINICAL LECTURE

ON CASES OF

MITRAL STENOSIS, AND WHAT WE CAN LEARN FROM THEM.

Delivered at the Central London Sick Asylum,
March 15th, 1898,

By A. E. SANSOM, M.D., F.R.C.P., &c.,

Physician and Lecturer on Clinical Medicine, London Hospital, &c.

GENTLEMEN,—I wish to speak to-day about cases of mitral stenosis and what we can learn from them, especially what we can learn from modern investigations.

In the first place we will examine this patient, a man at about middle life, to ascertain the physical signs, taking this as the type case. It is very important in such cases to commence with inspection, and indeed I would never omit it. Now if you put your eye down and look across his chest, you will notice a slight prominence in the heart region; the præcordium is convex, and the chest is more prominent on the left than on the right side. But the prominence is to the right of the nipple; that is important in connection with other signs. I do not find any decidedly marked apex beat, and I do not detect anything which I should interpret as a thrill, nor should I say that there is an abnormal pulsation evident there. Where is the maximum of pulsation? I believe it to be in the fourth left interspace, that is, over the right ventricle; it is the heaving of the right ventricle. Next as to percussion; percussion confirms me in the opinion that there is an absence of signs to the *left* of the nipple. An important point is to notice the angle which the heart makes with the liver. You can see there is a fulness here, and there is a prominence between the mid-sternal line and the nipple line. All the signs, then, are inside (right of) not outside (left of) the nipple line, which I think is most important. All show that the right ventricle is enlarged.

We will now proceed to auscultation and see what signs we have. The first thing which we notice is a very irregular rhythm of the heart. Occasionally I hear a loud second sound. The heart beats vary in force very much, but occasionally the second sound over the pulmonary valves is louder than normal. Now I hear a systolic murmur, and notice that it tails off from the first sound. Occasionally I hear a reduplication of the second sound, a double sound in the diastolic period of the heart. A systolic murmur is heard near the middle line, not away from it. It may be said that there is a systolic murmur at the apex of the heart. I do not agree with that, because I think it is to the right of the apex, and certainly in many cases of this affection I have found it to the right of the normal apex, that is to say nearer to the tricuspid than it is to the mitral orifice. Many people say that there is a systolic murmur of mitral regurgitation. But I should say pause, do not be too sure; think whether it may not be a tricuspid murmur. Some people say if you have got a tricuspid murmur you must have pulsation in the veins of the neck, or pulsation of the liver, neither of which occurs in this case. It is not so; you may have a tricuspid murmur when there is no pulsation in the veins of the neck, although there is murmur enough to show that there is some tricuspid reflux, it does not follow that the stream is sufficient to be causing any obvious backward flow.

What brought this about? First of all there is enlargement of the right side of the heart, and I have not got any evidence that there is enlargement of the left ventricle. There is a condition in which the left ventricle is small and the right ventricle is very much enlarged. In mitral insufficiency this does not occur; if there be any mitral insufficiency it is the left ventricle which is enlarged. In mitral regurgitation it enlarges more and more, and the physical signs show it towards the left axilla. Here it does not extend towards the axilla. What then are we justified in considering? That there are not the conditions of mitral regurgitation, but of mitral stenosis, because the left ventricle is not enlarged and the right ventricle is. The right ventricle hypertrophies because there is increased force required to drive the blood through the lungs and pulmonary veins into the [†] auricle, and from the left auricle into the left

ventricle; the obstruction is at the left auriculo-ventricular orifice. I do not think there is now a characteristic presystolic murmur, but there may be sometimes. A presystolic murmur is important but variable; you may hear it sometimes, but not at others. Now there is a circumstance which tells me there is mitral stenosis apart from the inferential evidence I have spoken about, namely, a sudden first sound. In mitral regurgitation you get obliteration of the first sound, the murmur drowns it and replaces it, but in mitral stenosis at certain parts of the præcordium you hear a snap—a tension sound.

Now we will go back and ask why this is. Why has it come on? If I were to ask the man whether in the course of his life he has had rheumatic fever he would say he has not, but that he had scarlet fever in 1896, and the scarlet fever was followed by rheumatic phenomena. Let us pursue that a little further. When I come to examine a number of cases of mitral stenosis and mitral insufficiency, I find in the mitral regurgitation cases that there is a large preponderance of histories of acute rheumatism, but in the mitral stenosis cases they do not show signs of acute rheumatism so much. In the case of mitral regurgitation there is a very pronounced endocarditis about the mitral orifice, and these patients may have repetitions of it. The result of that is that the valves and the cords and columns get retracted from one another, there is a considerable amount of change, and the auriculo-ventricular orifice is widened. But in mitral stenosis the evidences of rheumatism are much more slight, sometimes non-existent. There may be no evidence of rheumatism at all either from family history or from the individual himself. On the other hand, some of these patients have had scarlet fever. This man has had scarlet fever without any previous rheumatic history or tendency, but he got rheumatism afterwards. I have had many such cases in patients who have suffered from measles. Those who have had scarlet fever in that way are more likely to have mitral stenosis than those who have had acute rheumatism. I take it the reason is that the change in the valve that produces the stenosis is a much more slow-going one, much less pronounced, less extensive, much more local than that which produces mitral regurgitation. Well, in this case I could not say that this was not a rheumatic form of endocarditis,

only that it is the form of endocarditis which is not accompanied by very pronounced signs of rheumatic fever. You may say "Is it rheumatic at all?" I will come to that point presently. On that question I have very decided evidence that a thoroughly rheumatic case showing signs of mitral regurgitation may, in course of time, come to be a pure mitral stenosis. I have had several observations of this fact, so that I cannot have any doubt that the rheumatic form of endocarditis can eventuate as a mitral stenosis; the only thing, as I have said, is that it is probably of a much more slow-going character than that producing regurgitation. What is the effect of this? You have enlargement of the right heart, but no effect on the left heart. The man is weakly, and he has had bleeding at the nose, and has spat blood. Epistaxis and hæmoptysis are two of the frequent symptoms of mitral stenosis, whereas the gradual swelling and gradual oncoming of anasarca or œdema are much more characteristic of mitral regurgitation.

I want to show you another patient at this point. She tells me that she had rheumatic fever when she was between seven and eight years old, and she is now thirty-two. You see she is very thin, and I learn that she is not hearty in any way. This case is instructive in connection with the one we have just left. I do not see any marked prominence in the heart region here, and I do not get the signs of right ventricle enlargement that I did in the other one. She has a loud systolic murmur. You get the tap I mentioned just now, like a hammer hitting your ear. You have a murmur that is so often supposed to be one of mitral regurgitation, but it is heard towards the tricuspid area, so it is just as likely to be tricuspid as mitral regurgitation. Many of these murmurs in mitral stenosis that are supposed to be mitral regurgitation murmurs, and therefore to indicate a combination of mitral regurgitation and mitral stenosis, are nothing of the sort. The murmurs are those of tricuspid regurgitation, varying very much, always to the right of the place where a mitral regurgitation murmur is heard, not over the apex exactly; and if you put the stethoscope to the left you will hear this "tap," which tells you that the first sound is not accompanied by a murmur; it is a sudden membranous tension sound which seems to hit your ear. Then here we have something very decisive. We have the same evidence as in the other case *plus* something else.

The patient is ill-developed, and she has always been weak. That is because ever since that early time of her life she has had a small left ventricle, and there has been an obstruction practically all through her life preventing her getting the proper supply of blood to the aorta; she has had practically arterial starvation.

We have certain points to think about. In the first case we have no evidence of rheumatism except a post-scarlatinal rheumatism; here we have decisive evidence of acute rheumatism at an early age. In neither of these two cases at present have I got the typical presystolic murmur, but when I saw the patient before there was a typical presystolic murmur. I do not propose to go into the question of what a presystolic murmur is, but I will define it if you like as a murmur which begins in the diastolic period, however far back it may be; it sometimes goes through the whole diastolic period before the contraction of the ventricle, and then stops suddenly with a snap. In some cases it occupies the auriculo-presystolic period alone. In its typical form, however, it occupies not only the auriculo-systolic period, but the diastolic period, so that it is a diastolic-presystolic murmur. Now let us think why this was brought about. In this case I think it was brought about in the earliest days of childhood, the left ventricle being small, and the aorta ill supplied with blood. Abroad this would be characterised as a case of pure mitral stenosis. What do we mean by a pure mitral stenosis? We mean a condition in which, if we were to look at the orifice between the left auricle and the left ventricle, it would be seen to be in the form of a funnel with the narrowest part of its opening into the left ventricle. I have abundant evidence, cogent to my own mind, that it is rheumatic in causation in the vast proportion of cases, although it is a very slow-going form of rheumatism. In the second case I think we have got evidence in point. In France many people do not think it is rheumatic at all. Duroziez has emphatically said that it is not a rheumatic condition. Now, it is quite clear that it is not a congenital anomaly; we do not find it in association with congenital anomalies. It is always associated in early life with something rheumatic. In Potain's great book published three or four years ago, there are a large number of observations by Teissier, which

to his mind conclusively show that it is a tubercular affection. But to my mind and to that of many others that is not correct. He quite agrees that there is no tubercle in the valves or about them, but he says that sometimes the cases are associated with tuberculosis of the lung. That is true; sometimes they are associated with tubercular disease in the lung, but I think very rarely. Still I have seen in a woman with acute tuberculosis very pronounced mitral stenosis; but that does not bear out the reasoning that there is tuberculosis to start with. It seems to me that that cannot be proved by anything that is positive, and the explanation of tuberculosis being associated sometimes with mitral stenosis is that with a narrowed mitral orifice and small ventricle there is imperfect arterial supply and a want of resisting power and tone to prevent the invasion of tubercular disease. I think that is the reason why tuberculosis is found in mitral stenosis cases more frequently than in those of mitral regurgitation or any of the other forms of heart disease. Is there any other cause besides rheumatism? There is an exception to rheumatism being the cause in later life; then you see it in chronic renal disease. I have seen it as a consequence of rheumatism at all ages up to forty or a little over. In older people it is to my mind due to a slow fibrosis in association mostly with chronic renal disease, when probably endocarditis had nothing to do with it in the first instance. I have had abundant evidence on which to base this latter assertion, and some French writers have observed cases pointing to the same conclusion.

I have now come to a point which I think is very important from a physiological point of view. Supposing this were due, as I have said, to a slow-going endocarditis, how must that occur? In the funnel-shaped orifice we must assume that adhesion takes place between the curtains of the valve, that they get fused together, and the blood must, from force of circumstances, extrude evenly through the orifice, and in the systole there must be an even pressure in the ventricle below it. But if it occurs in that slow manner the original trouble must be at the edges of the valve. Granting that the adhesion was from the circumference, there must at one time have been an incompletely closed aperture. How then was it there was not regurgitation? There must have been regurgitation unless it was prevented by something. It could not be prevented

at the valvular orifice if the changes took place all round it and the orifice was thickened and made firm. What then did prevent the regurgitation? I think I can tell you. We have had a great deal of evidence on that point lately from Potain, who has gone over the whole question extremely carefully in the 'Clinique médicale de la Charité,' and from Dr. Samways of Mentone, who studied the subject very carefully in Cambridge, and did a great deal of experimental and very valuable research and statistical inquiry into the records of Guy's Hospital.

Now let us see how that stands in history. First of all, a great many years ago it was said by Dr. Dickinson that he could not make the murmur indicating mitral stenosis out as presystolic, because he felt with his hand that the murmur actually began when the apex beat of the heart was felt with the finger. He said it was not presystolic, but systolic. I happened at that time to be working at the cardiograph a good deal, and in some of my cases of mitral stenosis the diagnosis of which I had no doubt about whatever I found there was an enormous lifting up of that portion of the cardiogram that indicated the auricular contraction. Not only so, but the force of the auricle contributed to the whole elevation that we thought formerly to be due entirely to the ventricle. I found that in mitral stenosis there are some cases where the auricle cannot thus act upon the left ventricle, where there is a marked stenosis its force is broken. If the orifice were very narrow the force would be broken before it got into the left ventricle, but in some cases it is sufficiently powerful, not only to make the auricular rise, but also to contribute largely to the elevation of the left ventricle; and if it is powerful enough to do so it must be powerful enough to be felt by the finger. So I have said that the impulse of the heart, supposed to be caused by the left ventricle, was really, in a considerable degree, caused by the left auricle. That view has been taken up by Professor Potain, and proved in the most elaborate way. He has gone over the question, and in his article on the apex beat of the heart, and also in his lecture on the physical signs of mitral stenosis, he has exactly endorsed all this by the most precise investigation. He shows that normally the auricle contributes very considerably to the ordinary apex beat of the heart. What we

feel of the normal apex-beat of the heart is in part produced by the auricle, but still more so in stenosis, where the auricle becomes hypertrophied. Then there is something else that it does. If this observation is right, the auricle does not cease its contraction before the time of the contraction of the ventricle, but works with the ventricle. It continues on to the time of the ventricular contraction, and you must not forget that the cardiograms were taken not from intra-ventricular pressures, but by a machine which recorded the movements of the ventricle. According to Potain's investigations it is abundantly proved that the auricle carries on its contraction up to the time when the ventricle has acquired force enough to open out the aortic valves. Now you will see why it is that regurgitation is prevented in mitral stenosis. If there had been regurgitation there must have been dilatation of the left ventricle. If the regurgitation were prevented while the obstacle to the entry of blood into the left ventricle was maintained by stenosis, then the left ventricle would not be dilated, but would be smaller than the needs of the organism required, and that is what I think happens. You will remember that physiologically the strength of the left auricular contraction is kept up whilst the ventricle itself contracts, and until the moment of the opening of the aortic valves. In the early stages of mitral stenosis the great effect upon the auricle is one of hypertrophy resulting in increased force, and the consequence is that the auricle contracts and keeps contracted during the whole of the time until the ventricle has got free play into the aorta. There is no pressure back and therefore no regurgitation, and in pure mitral stenosis there is no regurgitation—it is all obstruction. That has been proved by the course of events. What happens at certain periods of the history, but more in the later periods, is that the hypertrophied auricle after a time becomes dilated and enfeebled. But then, as Dr. Samways has pointed out, that is in the later stages in the great majority of instances. Anything which depresses the nutrition weakens the muscular fibres, the auricle becomes incompetent to fulfil its task, and the heart begins to fail. Again, even in the later stages its failure must be very slow-going, because I have seen a left auricle as thin as a visiting card; I have seen it with layer upon layer of coagulated fibrin, and I have seen

it when there has been practically no muscle at all. The whole circulation was carried on without an effective auricle by the right ventricle driving blood through the lungs into the pulmonary veins, and from the pulmonary veins into the left ventricle.

One important point which I have heard as an objection to the view expressed of the auricle being able to maintain its contraction right up to the beginning of the ventricular contraction, is that it would assume that the auricle was able to overcome the force of contraction of the left ventricle in some degree. And so I think it is. If it is a question of mechanics, the contraction of a small sphere will sometimes and under certain circumstances overcome the contraction of a large sphere and be at a mechanical advantage. But there is something more than that, because I should think you cannot judge this from the mechanical conditions of a contracting sphere, or from mathematical data alone. You have to think of the auricle as having a different endowment as regards muscle at one part than it has at another. It seems as if the contraction of the muscle began in the pulmonary veins, and the blood was, as it were, squeezed onwards, the strongest force being applied at the last. That may explain why it is that the presystolic murmur is so loud just at the end, because the most muscular part which contracts is the auricular appendix. One objection that has been urged is, how is it that blood is not driven back forcibly into the lungs in such cases? I have heard it reiterated that there is no valve between the left auricle and the lungs. I beg to differ seriously on that point. There is a valve and a very important one which people forget, and that is the circular fibres in the pulmonary veins themselves, which exert a powerful sphincter action, which can be easily demonstrated, and that sphincter tends to keep the blood pressure from the lungs in the cases both of mitral stenosis and mitral regurgitation.

You will remember I said that sometimes there is a deceptive sign as of mitral regurgitation; a murmur is heard that we are apt to put down as mitral, but is really of tricuspid regurgitation. The great thing in these cases is to put before ourselves the following points:—Is the left ventricle dilated, or is there absence of evidence of anything like dilatation? Is the left ventricle smaller or larger than normal? Is there a disproportionate en-

largement of the right ventricle as compared with the left? If the enlargement is of the right ventricle, and not of the left, and if there is the snap of the first sound, which means that where that is heard the first sound as produced by the left ventricle is not accompanied by a murmur, then I think we have evidence that there is a mitral stenosis not accompanied by mitral regurgitation. The history of that affection is a different history to that of mitral regurgitation, it is a different entity altogether and requires separate study. I think every year we have been having more clear and decided insight into what that morbid condition is.

Toxic Infection or Ptomaine Poisoning.—

One factor which is frequently overlooked in the treatment of children is toxic infection or ptomaine poisoning. One is frequently called to attend a child, and finds the little one presenting these symptoms:—Mild delirium. Temperature 103° to 104° , pulse rapid and hard—120 to 140. Tongue coated and breath foul. Skin dry and hot; face presenting a tired expression, especially noticeable in the eyes. Vomiting and pains over the abdomen; usually constipation. On questioning the parents we find the child has complained for some little time, has had slight headache, not much of an appetite; has been cross and peevish, and has had "stomach-ache." The fever in 50 per cent. of the cases is ushered in with a chill. If a case of this nature happens in a district where there is more or less malaria the practitioner diagnoses it as such, and prescribes quinine, and then is quite surprised that the condition does not yield to his specific treatment. In twenty-four or forty-eight hours after seeing the patient he begins to think strongly of typhoid, as the symptoms begin to resemble those of that disease. In reality they are due to ptomaine poisoning, toxic infection taking place in and from the colon and possibly intestines. Make a careful examination by exclusion. If possible examine the blood for the malarial parasite, and use Widal's test for typhoid. In all probability both examinations will give negative results. While doing this, place the child on lime water and milk, half and half, every four hours. Give albumen water every two hours. For medication—

R. Hydrarg. Subchlorid. .. 1 gr.
Sodii Bicarb. ... 3ss.
M. et ft. Pulv. vj.

Sig. One every hour.

With the last powder, one to three grains of Rochelle salts, according to age. Continue the restricted diet for a few days. We believe the condition described is frequently wrongly diagnosed and mistreated.—*Charlotte Medical Journal.*

WITH DR. ABERCROMBIE IN THE WARDS OF CHARING CROSS HOSPITAL,

February 10th, 1898.

LADIES AND GENTLEMEN,—This is a young man 21 years of age, who was in this hospital under my care seven years ago. His history then was that he had been ill about four months with occasional diarrhoea and some degree of abdominal discomfort. His abdomen was extremely hard, especially in the right iliac region, where there was some fulness. Examination *per rectum* showed that there was a firm swelling coming down on the right side of the pelvis, and Mr. Boyd and I agreed that the case was one of appendicitis and suitable for operation. When the abdomen was opened it was found that he had tubercular peritonitis; grey granulations and small yellow caseous nodules were found freely scattered over the peritoneum. The resistance which had been felt in the right iliac fossa was due to adhesive inflammation. The peritoneum was therefore flushed with a solution of mercury, with some iodoform added to it. There was a great deal of fever after this. I treated him first with iodoform internally; but as he did not improve, and his temperature remained high, after a few weeks I decided to try mercurial inunction, and in a very few days after that the temperature became normal, and he showed marked improvement: but this progress was not long maintained, and his condition remained stationary until about four months after the operation, when an abscess discharged itself close to the iliac fossa, and a good deal of foetid pus came away; from this time he rapidly improved, and soon was practically well. The other day he came in for what might have been appendicitis again, namely with a history of pain and tenderness in the right side of the abdomen, and some constipation, and he also had a very tender swelling in the right iliac fossa. All the tenderness subsided very soon, and the induration which was at first felt is hardly to be made out now. I thought it would interest you to see a case of undoubted recovery from tubercular peritonitis, the patient having remained free from manifestations of tuberculosis for so long a period. I may say I believe

tubercular peritonitis is eminently curable; it is a disease about which I would never give an unfavorable prognosis if I could help it, especially if the case be seen early. There is nothing acts so beneficially on the disease as the ung. hydrarg., either rubbed into the abdomen or applied on a flannel belt to be worn for an hour or so daily.

I have seen him several times since, and know that he has not had any return of his tubercular peritonitis, though you can feel that there must have been considerable matting. In these cases of appendicitis one must be careful to avoid anything approaching constipation; a slight degree of this may determine an attack of appendicitis.

Our next patient (æt. 29) is one about whom it is not so easy to speak as positively as the last. He has been a soldier, and has served in India, from which country he returned three years ago, invalided for dysentery. He went to Netley and got, as he considered, perfectly well. That was in May, 1895. In November of that year he was admitted to this ward, under Dr. Bruce, with an enlarged and tender liver. Dr. Manson saw the patient with him, and both thought that the enlargement of the liver was due to tropical abscess. Mr. Morgan accordingly punctured the liver in six or seven places with a trocar and cannula in order to try and hit off the abscess, but he was not successful in finding any pus. After that the patient was put on iodide of potassium, and in the course of six weeks or two months he left the hospital greatly improved. He tells me, however, that he has never been quite well, and has not since been able to do hard work as he could before his illness. He came back here three weeks or a month ago because his legs were swollen. That is probably due to albuminuria, as albumin appears in his urine in considerable quantities. The interesting point about him is his liver. On physical examination we find it is almost exactly as it was before, enlarged in a peculiar way, not downwards but upwards, so that you do not feel it lower than usual in the abdomen. The upper border of the liver is on a level with the fourth interspace, and the upper limit of dulness maintains the same level round to the spine. The dulness being so high as this raises the question whether the patient is suffering from a pleuritic effusion, a suggestion which is negated on further

examination. As far as I know, there are only two conditions that tend to enlarge the liver upwards: one is abscess of the liver, commonly situated in the right lobe; the other is hydatid cyst of the right lobe. I think it is natural that the first thing to suggest itself in view of his having suffered from dysentery in India is abscess of the liver. Dr. Manson, our lecturer on tropical diseases, was kind enough to see him, and he thought, as he had done two years previously, it was probably a case of encysted abscess of the liver. With a long exploring needle we punctured the liver in two places, but we did not find any pus. When he was punctured on the former occasion he derived very much relief from it, and that would seem to be the case also now. He has had some irregular fever since he has been here, as you will see by the chart. Since the puncture, with one exception, there has been a steady subnormal temperature. There is another cause at work which may possibly account for his being better, and that is we put him again on iodide of potassium. His spleen is very considerably enlarged, although it cannot be felt, but there is a large area of dulness at the left base, which I think there is no doubt is due to enlargement of the spleen. As you see, the patient is very anæmic, and he has slight clubbing of his finger ends. That clubbing made us think of tuberculosis, but we have examined him several times from that point of view, and have tested his sputa for tubercle bacilli, but there is no evidence that he has tuberculosis. The patient having improved so much on iodide of potassium raises the question whether this is a syphilitic liver. I think our knowledge that the liver is the same size as it was two years ago precludes the idea that he is suffering from syphilitic hepatitis. This disease would lead to contraction of the organ in that time, and there would be signs of cirrhosis of the liver of the ordinary kind, and I think this patient would now have had ascites.

I am still inclined to regard the case as one of hepatitis in association with his dysentery, and quite possibly there is an encysted abscess in his right lobe. At the present time I propose to push the iodide of potassium, and thus give it a fair trial. There is no history of his having had any symptom suggestive of liver affection while in India.

The next patient is a boy with adhesive peri-

carditis. He is 16 years old, and, as you will see, he is very small for his age. He came here in January last year with an extreme degree of bronchitis and some emphysema of the lungs, and a good deal of ascites and anasarca of the legs. I thought at first the case was one of emphysema and dilated heart, with acute bronchitis supervening. After a little while he developed right pleural effusion. That subsided, and then I noticed that he had a very big liver; we could not feel this at first, and he was too ill to be examined. His condition reminded me of that of a boy with mediastino-pericarditis, who was ten years old when he died, and had been under observation two years, his illness having dated from a little time anterior to that. I show you the specimen from our museum, from which you will see that the pericardium is extremely thickened and almost cartilaginous. Both layers of pleura were adherent to it outside, and the pericardium is adherent to the sternum. As a result of that extreme thickening of the pericardium and its fibrosing condition, it keeps on tending to contract instead of yielding, and so the growth of the heart is arrested, and owing to the mechanical impediment to the return of blood into the heart, enlargement of the liver, ascites, anasarca of legs, and cyanosis result. I began to think that this boy might be also suffering from this peculiar fibrosing pericarditis. He has never had any heart murmur at all. The area of cardiac dulness is practically obliterated by emphysema, so that we cannot say anything about it. His pulse is always extremely small and weak, and anasarca and ascites have been present all through his illness. Before the ascites was so marked we satisfied ourselves that the liver was large and smooth and firm. It presented one peculiarity, namely, that it fell away from the diaphragm, perhaps because the suspensory ligament had got stretched. This boy has always had a certain amount of orthopnoea, and sometimes we have noticed that he has decided cyanosis. After a time the anasarca became so great that we had to deal with it, and incisions were made into his legs under antiseptic precautions and the legs were drained. The ascites was for a time relieved by this, but subsequently we were obliged to resort to paracentesis. Paracentesis has now been performed thirteen times; Southey's trocar and cannula and tubes have always been used, and the quantity drawn off has varied between 143 ounces

to 250 ounces, at first about a fortnight intervening between the operations, but latterly the interval has been much shorter. The need for the measure comes on sometimes very suddenly, as a rule the degree of dyspnoea being the determining indication for the operation. The fluid drawn off lately has been less, probably because he is not strong enough to stand so much pressure from the fluid as he used before having it drawn off. His general condition is fairly satisfactory; he takes his food well, and we provide him with changes to tempt him. You will see in his legs besides the scars of the incisions (incisions are better than punctures for this condition, because a puncture will often heal up before the requisite amount of fluid has come away), the peculiar condition of the skin caused by the anasarca; it is thickened, and cannot be pinched up. I have noticed this state in several cases after prolonged oedema. There is a good deal of ascites, but the abdomen is soft. You can now feel the liver a good deal enlarged, but I do not think it is as big as it used to be; these chronic "nutmeg" livers do contract. I ask you to listen to his heart, and I think you will agree that there is nothing about it to make any one think that the case is one of heart affection. Sometimes I have fancied that I could hear some friction râles over the præcordial area, which might be expected from the adhesions taking place extra-pericardially to sternum and pleura.

The causation of this disease is extremely obscure, and the cases are rare. There is a certain association with tuberculosis; that is to say, in some of the cases the mediastinal glands about the bronchi and trachea, or sometimes glands in the neck, have been found caseous, but I am disposed to regard the association as accidental rather than one of causal relationship. As far as I know this affection is very much commoner in males than it is in females.

The next patient is a woman aged 50, who has been in the hospital about a fortnight, and had been ill for some nine weeks before that. She complained of shortness of breath and pain in her chest. Three days before she came in, the doctor who was attending her tapped her left pleural cavity and drew off three pints of blood-stained serum. Since she has been in we have twice had to relieve the dyspnoea by tapping the chest, and each time we drew off a pint of fluid. I show you

the fluid; it is a thick, chocolate-coloured fluid. There is a slide of it under the microscope. You will see, in addition to altered blood-corpuscles, numerous large epithelial cells undergoing granular degeneration. Hæmorrhagic effusions are not very common. I think at her age the most probable diagnosis would be malignant disease of the pleura. Is this primary or secondary? Secondary carcinomatous affections are much commoner than primary. As far as we know, however, in this case there is no sign elsewhere of any primary disease. Generally such a condition is secondary to carcinoma of the mamma. The fact that the effusion is unilateral would make me think it is not secondary to carcinoma anywhere else, because if it were the new growths ought to be disseminated in both pleuræ. One is inclined to make the diagnosis, by exclusion, of carcinoma of the pleura certainly and lung possibly. She is too ill to permit of an exhaustive examination. As regards physical signs, before tapping her there has been absolute dulness over the left side, which is only partially modified by aspiration. Yesterday we examined her shortly after the tapping, and then the first two spaces were not so dull as they had been. On examination there has generally been bronchial breathing towards the inner end of the upper spaces, and that I think may be due to enlarged bronchial or tracheal glands. These glands are apt to be secondarily enlarged, and may conduct sounds direct from the trachea to the ear. It is a kind of tracheal breathing rather than bronchial. The heart is not very easily to be felt, but when we have felt it it has been to the right of the sternum, and it has not gone back since the tapping. That is capable of more than one explanation. It is possible that it might be held there by pericardial adhesions, or there may be some growth in the mediastinum pushing it over; this is rendered probable by some curious attacks of dyspnœa she has had. After the former tapping here she has had two or three serious attacks of dyspnœa, which were relieved by nitro-glycerine. The dyspnœa may be due to pressure by a mediastinal tumour, but I must add that most of the signs of mediastinal tumour are absent.

There are other sources of hæmorrhagic effusion into the pleura, and in a young person I should have first thought of tuberculosis. It is not common, and has only come under my observation three times. Once the patient was a child who had

a small effusion at one base. This was punctured, and we drew off five ounces of blood-stained serum. The fluid re-accumulated, and subsequently became purulent. This was treated in the usual way, and he got well. He came back a year afterwards with tubercular peritonitis, so that it is fair to assume that the effusion was associated with tuberculosis. In the other two cases the events were different. They were cases of empyema, in which an opening had been made and a tube inserted, and hæmorrhage took place some days after the tube was put in. The blood was first discovered on the dressings. On both occasions it was practically impossible to stop it, and in both cases the pleural cavity was found to be full of blood, and a large area on the surface of the collapsed lung was found to be ulcerated. In both these cases there had been tubercular ulceration breaking down on the surface of the lung. There is yet another source of hæmorrhage into the pleura, which might be put out of court in a child, but not in this patient, and that is aneurysm. I had a case last year in a male patient twenty-six years of age, who came in for pleural effusion. I was surprised on making an exploratory puncture to find a very highly blood-stained fluid. His dyspnœa subsequently increased and we had to tap his chest, when the fluid that we removed consisted almost entirely of blood. The pleural cavity filled up very rapidly again, and it was subsequently shown that he had an aneurysm of the descending aorta leaking into the left pleura; I believe that is the most common result of aneurysm of the descending thoracic aorta, that it should leak into the left pleura. In a patient aged twenty-six aneurysm had not suggested itself to me, and I was unprepared for it. He was said to have had a history of heart disease, and that heart disease might have meant that he had a murmur due really to the aneurysm, but conducted and heard through the heart.

Our last patient is a young woman 21 years of age, who comes in with paralysis of the left foot. The causation of it, however, is very difficult to decide. Eight weeks ago she had some pain in the leg and swelling of it, but says that her general health was good throughout. I have tried to make out whether she has had an attack of enteric fever, but there is no proof of it. Of other causes of paralysis such as this, one thinks of

alcohol, but I never knew alcoholic paralysis to be unilateral. No doubt these cases are sometimes associated with tuberculosis, but there is no evidence in our patient of tuberculosis. Therefore, I suppose we shall have to fall back on the assumption that it is a post-influenzal condition, which really amounts to saying we do not know what the cause is. When she came in she had complete paralysis of the extensor group of muscles of the left leg,—that is to say, the tibialis anticus and the extensors of the toes,—and she could not move the foot in the least. She had also an increased knee-jerk on this side, and a very slight affection of common sensation in the leg. When tested electrically it was found that the affected muscles presented the reaction of degeneration. She has improved since she has been in the hospital, and she can now distinctly move her toes. I think any improvement in these cases, no matter how slight the change may be, is always a most favorable sign, but until there is evidence of improvement it will be wiser not to say much on the point of prognosis.

Now as to the pathology of these cases. Twenty years ago this would have been labelled, without doubt, a case of subacute anterior poliomyelitis,—something halfway between infantile paralysis and progressive muscular atrophy. Ten years ago we should have labelled it certainly peripheral neuritis. Now I think opinion is again changing round, and we are getting very sceptical (I have myself been sceptical for a long time) about peripheral neuritis. I admit the possibility of it in leprosy, and also as a result of supposed cold, but in these cases you always have marked sensory phenomena; in neuritis from cold, for instance, there is often extreme pain. I think if this were a local neuritis we ought to have much more marked sensory phenomena. Latterly opinion has been coming back from peripheral neuritis to central lesion as the cause of the symptoms. The view that is now taken of these cases—and in this matter we are greatly indebted to the work of my colleague, Dr. F. W. Mott, F.R.S.—is that you must regard the nerve-fibre as only part of a system, that you must connect it with the cell in the anterior cornua, and regard the cell with its processes and prolongation into a fibre, as an entity called the neuron. If anything happens to the cell, the effects will be shown first at the most distant point, the periphery, and that is why some of these cases have shown on microscopical ex-

amination changes in the nerves which have been called peripheral neuritis, and perhaps nothing has been recognised centrally, the fact being that there was a poison acting on the nerve cell, which produced its effects and betrayed itself by degeneration at the most distant point, and so it appears in the extremity. What the poison is in this particular case we cannot say. It may be that some of the anterior cornual cells have been poisoned so deeply that they will atrophy; but others can be made to take up their work, and I hope that with assiduous treatment we shall see her get quite well.

Ascaris lumbricoides.—The general practitioner, as a rule, writes Fuente, attempts to make a diagnosis without microscopical examination of fæces. The first symptom generally is sudden acute abdominal pains, and the history then shows that slight pains have been experienced before, but, being transient, were not complained of; or the fact may be elicited, if dealing with an infant, that it has lately been restless, crying a great deal, refusing nourishment, &c., which condition, however, passed off within a few hours or a day. These pains are characterised by difficulty experienced in determining exact localisation, and it is not rare to find a more or less tender point on pressure over the region designated. Very young children, who permit palpation of the whole abdomen without resistance, suddenly start and cry when a certain point is touched, and this is repeated each time this portion is reached.

The differential diagnosis between inflammatory processes is established by the absence of high fever. A slight rise of temperature may occur toward evening, but during the day the patients present a perfectly normal temperature. In exceptionally bad cases the pain is so great that older children moan and cry out. Movements of the bowels are always present; either there is a diarrhoea, or quite as frequently the passages are perfectly normal, this being a valuable diagnostic sign. Convulsions have been observed, due to presence of ascarides. The sudden, nearly epidemic appearance at times of ascarides in certain localities present a peculiar phenomenon. Exceedingly remarkable is the enormous contraction of the field of vision in cases where intestinal parasites are present. In some cases this may be demonstrated by a very superficial examination.—*Monthly Cyclopædia.*

THE SURGICAL AFFECTIONS OF THE STOMACH, AND THEIR TREATMENT.

A Course of Lectures delivered at University College during February, 1898, by

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No. IV.

GENTLEMEN,—The last time we met we were considering the actual operations which were performed for gastric ulcer, or gastroraphy, and I told you about the preliminary incision which is made through the abdominal wall in the middle line above the umbilicus, and that it may be supplemented by a transverse incision in certain cases where it is necessary to obtain more room. The transverse incision is hardly necessary in most instances, but in two of my own cases it was required, owing to the awkward situation of the ulcer. Fortunately for us, and for the patients, the majority of ulcers are situated on the anterior wall of the stomach. It is quite possible to have all the symptoms of perforation by gastric ulcer—sudden onset, intense pain, collapse, distension of the abdomen, the dulness, the rapid pulse, effusion of serum, and acute peritonitis—when no actual perforation can be found. Those cases will always be a source of embarrassment to us. They are supposed to arise in this way:—that the ulcer has approached from within, very nearly to the surface of the stomach, and the ulceration has excited inflammation on the peritoneal coat of the latter, which inflammation has spread to the rest of the abdominal cavity. I have told you apropos of other points that through a damaged wall of any part of the alimentary tract septic matter may escape without actual breach of surface—that is, that organisms may pass through a damaged bowel, whereas they will not pass through intestine which is intact, and in one of my own cases, the last one I did, no perforation was discovered, although there was extensive peritonitis and all the symptoms of the condition. And not only

was no opening found during the operation, but afterwards at the autopsy, although the ulcer was there, well marked and almost patent. Of course such a stomach will still hold gas and water very well.

Now, when you have made this opening, the escape of gas as well as of the peritoneal fluid will very considerably facilitate your examination of the interior of the abdomen. You can manipulate with far more ease under these circumstances than is possible in a healthy abdomen, and one notices this especially in abdominal sections on young people whose muscles are strong and irritable, and, as a rule, the intestines have a tendency to bulge out at once the moment the opening is made. After the escape of gas and fluid after perforation this is not the case. The next thing you do is to look for the ulcer. When found the stomach is squeezed upon and emptied gently through it. If there is any difficulty in this you must insert a tube through the opening, and squeeze out the contents of the stomach through that. Or, again, you may, as has been done, put a tube through the œsophagus and, with a funnel, pour water into the stomach, allowing it to escape through the perforation. In any case it is most important that the stomach should be emptied, otherwise when you are suturing it, and after you have cleansed the peritoneal cavity, fluid will run out, infecting your sutures and the remainder of the abdomen. You may wash the stomach out with plain boiled water, which is, perhaps, the best of all, and put in it at the end some brandy, which is a good antiseptic and a stimulant to the patient. Then the abdomen has to be cleansed before the suture is inserted, so that the latter may not subsequently be soiled. The cleansing may be carried out either by flushing or by sponging with the hand. This is a matter upon which surgical opinion is divided, namely, as to which is the most desirable way of dealing with it. You are aware that in many cases fragments of food have found their way into the peritoneal cavity, and there is a quantity of peritoneal fluid besides. How best are you to get this away? In my own cases I have lately used a sponge, though I was an ardent advocate of flushing of the abdomen in former years. At that time I had specially made two 2-gallon cans fitted with three taps each for flushing out the abdomen. The reasons for the latter

practice would be these :—In certain cases in which the condition has lasted for some time you have extravasation practically all over the abdomen. When the perforation occurred the patient was very likely in the vertical position. The immediate effect of the accident is that gas escapes into the abdomen. That gas, of course, has a tendency to rise to the higher part of the cavity, and there will be a tendency for the liver to be dragged down by its own weight, aided by the forcing of the gas above it. As the liver is partly suspended by atmospheric pressure its weight will suck the air between the diaphragm and the liver as it falls. And, besides, with that extravasation of gas there is probably extravasation of fluid into those parts—the subphrenic spaces on either side. Then on account of the pain and shock the patient will lie down, which again helps the diffusion of the gas and fluid, and his suffering will cause him to turn from side to side, allowing the fluid to get into the flanks and all round both kidneys and the spleen. The fluid will also almost certainly find its way into the pelvis; in two or three of my own cases I have found the pelvis full of peritoneal fluid and fragments of food, and to dislodge such matters from an irregular cavity is by no means easy. As I have said, in my earlier cases of abdominal section for septic conditions I used a three-way can, inserting one rubber tube into the pelvis, another into each flank, and turned on all three taps at once. Then there was a stream flowing from these parts towards the abdominal opening. The object of a simultaneous flushing of different parts is obvious, namely, that if it were done separately the septic matter would be simply driven in part, at all events, from one place to another without being evacuated. In order to thoroughly wash out the peritoneum, there really should be four or five tubes employed, so as to clear out the subphrenic spaces at the same time as the pelvis and flanks, the stream from all being towards the central incision. The objections to flushing, although theoretically it may seem very proper, are these :—it is imperfect in the first place. The stream does not penetrate into all the spaces between the coils of intestine, and carry away all the injurious material. The next is that it almost invariably leaves a quantity of fluid behind; and it is very difficult to remove all this subsequently, and what is left

behind is very prone to be septic, the water having mixed with some previously decomposed matter. Again, the flushing is liable to produce shock unless the water is carefully regulated to the proper temperature, namely, about 105°. If it is hotter it produces shock; if it is cold it produces greater shock. Then it is not possible to cleanse the whole abdomen without using a very large quantity of fluid—even gallons. Now that is all very well if you are employing water or normal saline solution, but it is not antiseptic, and the residuum in the abdomen is probably injurious to the intestines. But if you use a strong antiseptic it is dangerous from its toxic effects, and by its irritation it may produce increased shock. That is to say, if the fluid is strong enough to be antiseptic it will be injurious; and if it is not strong enough to be antiseptic it is of very little use at all, except mechanically to dislodge foreign matter, and this I think it does imperfectly. It is for these reasons that some of us adopt the method of cleansing the abdomen with the hand by the aid of sponges, and because, as I have pointed out, it does not follow that though there has been extravasation of the fluid through the gastric ulcer it has yet reached every part of the abdomen. It has been pointed out that in perforation producing effusion in front of the omentum and the transverse colon, the fluid is frequently dammed back by the latter with the omentum. Now, if you flush in such cases you distribute the fluid into parts which otherwise it probably would not have reached, whereas if you wipe the parts with sponges you still limit the effused material to the part to which it was originally extravasated. Again, you can pass soft perfectly sterile sponges with the hands or a sponge-holder into any part of the abdomen, and suck up the fluid by this means more rapidly than by flushing. And remember this, that in many cases you have to sponge the abdomen whether you flush or not. Therefore for these reasons I do not now flush. In one or two cases, where the intestines were partly through the wound, I gave a dash of hot water externally to wash away the débris. But if the sponge is employed it must be used methodically, or else some part of the abdomen will be missed. Now the course I should recommend you to pursue, and I think it is that which would be advised by most surgeons, is to give your attention first to the subphrenic spaces. Take a

flat sponge and insert it between the liver and the right side of the diaphragm close to the falx. Pass your sponge up and wipe outwards down towards the kidney on the right side. You will be surprised to find the sponge covered with lymph, and perhaps traces of food will be on it. If it is so you pass a fresh sponge in, and presently you will find you have left the surface of the liver relatively clean. It is well for you to leave a flat sponge in that situation until the end of the operation. Then the same is done on the other side between the liver and the diaphragm, and special attention ought to be paid to the region around the spleen, wiping it very clean and leaving a sponge in there too. Then, if you find as you get towards the back of the abdomen that there is fluid in both flanks, you may proceed to clear that out in the same way; perhaps you will find it better to use the sponge-holder for the fluid around the kidney. If you find there is debris of food, you will have to be extremely careful to get it away, and anything which may tend to become a focus for inflammation. If you have reason to suppose, as was the case in some of our patients, that the pelvis is full of septic fluid, you must slip your hand down over the intestines and mop it up behind the uterus and Douglas's pouch, even though the procedure takes some time. I have also found it a useful practice in cleansing the flank to leave a sponge on a holder or on a thread while going on with the rest of the operation. A flat sponge is also left over the intestines which appear in the wound while you are proceeding to suture the perforation; that will prevent the possibility of fluid from the stomach deluging the spaces which you have just cleaned.

The treatment of the ulcer is various. Some surgeons excise its base before closing the opening. This of course secures healthy tissue for the sutures, and cures the ulcerative condition at the same time. Of course, if you do not excise it you save time in the operation, but the ulcer may subsequently perforate or bleed, and thus destroy the patient's life, as happened in one of my cases. The patient recovered completely from the operation, and was in an excellent condition on the tenth day, but at that time he began to vomit pure blood, and eventually bled to death. Had I been able to excise the ulcer he would have been cured of the perforation at the same time, if he had got

over the operation. Of course, whether he would have survived the long operation due to the tedious process of getting rid of the indurated base of the ulcer is another story. The excision of the latter, as I have said, takes a considerable time, and leads to increased loss of blood, and for this reason most surgeons are against it, especially as the method of simple closure of the opening has been found to succeed in a great many cases. But of course such patients will require far longer after treatment in order to allow the ulcer to heal than had the original seat of the disease been excised.

As to the actual closure of the perforation, most surgeons simply insert one or two rows of interrupted or continuous sutures passed with very great care through the serous and muscular coats, and picking up a layer of very strong areolar tissue which lies under the submucous coat. I call your attention to the last point particularly. If your stitches do not include this fascial layer you will not have such a grip of the parts, and your sutures are a little likely to cut out. This is the most important part of the whole surgical procedure. You begin it an inch or so from the edge of the ulcer, and pass one stitch through these coats into the submucous tissue and out again. The next one will lie a quarter of an inch nearer the perforation, and this will produce a furrow. When a series of such sutures are drawn together the serous surfaces will be approximated point to point, leaving a groove in which the ulcer will lie, and the groove is closed at either end by two terminal sutures. Of course you may employ Gussenbauer's method or Czerny's. Or you may use a continuous suture; but this is not so convenient, because you want to have control over the whole of the opening in the stomach up to the last moment, so as to keep it clean before it is closed. If you are making your loops by continuous suture you approximate the serous surfaces piece by piece, and what you have previously closed may get septic before you have completed your stitching. But in putting in a second row of stitches most surgeons recommend a continuous line of suture. Undoubtedly the latter will save time. I have only used one row of sutures, and I believe the results have been as good *quâ* closure as those of other surgeons who have inserted two rows.

The sponges are now taken off the intestines,

the two from under the diaphragm are withdrawn, and those from the flanks are pulled up. We have by this means a relatively clean abdomen. But we cannot trust to it being in a perfectly aseptic condition; we must provide for a certain escape of fluid afterwards. Moreover, besides arranging for the escape of peritoneal fluid from the localised peritonitis which must follow, we have to provide for the possibility of the ulcer bursting out again, through our stitches giving way, and a second extravasation taking place. Therefore it would be most undesirable to close the abdomen straight off in these cases, as we do in many other abdominal cases, without drainage. Perhaps the best plan is to use strips of sterilised or iodoform gauze. What we have done in the hospital has been to pass a strip of this fabric upon either side through the central wound, one to lie between the diaphragm and the liver on the left, and another on the right, one going on either side of the ulcer; another can be directed towards the flank and the spleen on one side, and towards the liver on the other. Another strip would lie over the opening in the stomach, and if extravasation took place through giving way of the stitches, such packing would prevent the wider extravasation of the contents of the organ subsequently. But before putting in those it is wise to adopt a measure which is very widely resorted to now, namely, making a dam round the opening with the omentum. You can gently draw up this structure from its attachment to the transverse colon, and pack it round the line of suture in the stomach and over it. It very soon adheres to the latter, and makes an effectual barrier to subsequent extravasation. Then put in your packings and partially close the wound, close the transverse cut if you have made one, and leave the lower part of the central incision open. Then the loose antiseptic dressing usually employed in this case is put on and firmly bandaged. There is nothing like firm bandaging in these cases where the patient is suffering considerably from shock. They want all the blood they can get in their central organs, and pressure on the abdomen by pressing on the abdominal veins sends a good deal of blood into the heart, lungs, and brain. And in doing all this every second of time must be saved by every means. We must have all our instruments and everything ready at hand. As to whether the abdomen should be freely drained or not by tubes,

that is an open question with many surgeons. Some have thought that in view of the fact that extravasation is very likely to have taken place in the pelvis it would be better to forestall any trouble on that account by making an opening on either side in the linea semilunaris above the pubes or in the middle line, and to put in a drainage-tube. That has been done successfully, but I do not think it is necessary. In one case I made an opening under the ribs, and put in a tube which ran up between the diaphragm and liver behind the spleen, so as to drain the place where infection is very likely to be severe; but I was convinced from watching that case that the tube was of no use whatever; little or nothing came out of it, and what fluid was there would just as easily have been sucked up by a strip of gauze through the anterior wound. Moreover my feeling about these cases is that if there is going to be profuse septic peritonitis, you cannot drain it thoroughly by any means, and that has been the experience of a great many. The patient will succumb in such cases whether you like it or not. It adds to the severity of the operation, it increases the loss of blood, and it leads to loss of time, and I think surgeons are coming round to the belief that they save their patients without it, or not at all. The case in which I employed it myself was a successful one, but I did not attribute the good result in any way to this more complete drainage.

In those cases where the stomach is held fast by adhesions to the liver, diaphragm, spleen, or abdominal wall you may not be able to pull it sufficiently far forward to suture the opening in it, or the hole may be in such a situation that you cannot reach it. What then are you to do? More than once it has been treated by thrusting an apron of omentum into it and securing this by a stitch or two. Or the opening has been packed round with gauze and a drainage-tube put in just close to the opening, in order to carry off the contents of the stomach out of the abdomen. And success has sometimes followed in such cases. I think Mr. Godlee had a case of that kind. In his patient the perforation was there, but he could not get at it, and I am under the impression that the patient survived.

Now as to the subsequent treatment of these cases, that is a most important matter. The management of the wound is a simple affair; we

leave everything intact as long as we can. There is no reason to change the dressings for the first four or five days, if the patient survives as long, because the gauze has acted as a drain, and we want the part to remain undisturbed as long as we can for adhesions to take place. After that you will have to attend to these wounds, and this is a matter which might not, perhaps, occur to you in relation to packing with gauze in any case of abdominal section. At first the gauze is soaked with serum, and that is followed by lymph. The lymph gets into the interspaces of the gauze. So far so good. But presently the lymph will begin to organise. If it organises partly in the gauze and partly outside you have the fabric fixed to the peritoneal surfaces, and the adhesion is so strong that you cannot tear it out without some risk of displacing the viscera, and you cannot draw it out without bleeding, because vessels begin to form in the organised lymph comparatively early, and if you leave the dressing more than four or five days it will be found to be firmly fixed, and its withdrawal may increase shock; and if this cause the patient to vomit the straining may break down the adhesions, on the firmness of which the patient's life depends. It is not a bad plan first of all, in many of these cases where you have to put in much packing, to first throw over the wound an apron of gauze, and then stuff your strips in all directions into it, gathering up the corners in the way I show you. When you come to remove the drains you can pull out the strips of gauze, and that will loosen the surrounding apron. If you put zigzags in, and they hold tightly, it is a very serious matter to dislodge them. But if you pack in loose gauze into the apron lining the cavity, and keep the lips of the wound from closing, you will find very little trouble afterwards.

General considerations will guide us in the treatment of the wound subsequently; it is probably ultimately an aseptic wound, and no further trouble comes of it.

The general treatment of the patient immediately and remotely is the next important matter. Shock will probably be severe. You cannot do better in those cases than to elevate the foot of the bed and put on a firm bandage over the abdomen, and then administer an enema of hot water, about six ounces at 105°. In the first injection or two you can put half an ounce of brandy, and throw it up as high as

possible. That usually has a very good effect upon the pulse. This may be repeated every hour until the rectum begins to be irritable. Of course hot bottles for warmth and perfect quiet should be insisted on. At the same time strychnia in small doses under the skin will act as a very considerable stimulant to the heart and the system generally, and is most beneficial. Others also use camphor and digitalin hypodermically apparently with good results. But we are not so much in the habit of employing these latter as are surgeons abroad.

When the patients are recovering from the shock you must remember that they will want all their forces for repair, but we are debarred from giving the stomach much to do by the condition in which it is, and so we must feed by the rectum. We must take care that the rectum is treated with the utmost gentleness by the nurse, because if it once gets irritable your sheet-anchor is gone, for you can then feed by neither the mouth nor bowel. Therefore do not have too much brandy put into the rectum in the first instance, because after one or two injections it will cause irritation. Suppositories or the nutrient enemata are also a great help in these cases. Perhaps the best way is to alternate the suppositories with the injections, using the latter every four hours and a suppository in the intervening two hours; then what is left of the latter will be diluted by the injection, and if not absorbed it will be when the next injection is thrown up or expelled. Moreover when the suppositories are diluted in this way they are less likely to be irritable. The question then arises, when can you feed by the mouth? After the first twenty-four hours I think it is safe to introduce small quantities of fluid into the abdomen. Adhesion takes place very rapidly in the line of suture. When we have had occasion to open the abdomen again six or eight hours after certain abdominal sections we have often found enough adhesive inflammation to hold the parts together firmly. If we put a little brandy and water into the stomach in two-drachm doses with a little egg albumen, the stomach is quite able to deal with it, and being a non-irritating fluid it passes through the pylorus unchanged and gets into the duodenum very rapidly. If it were irritating and indigestible it would be retained by the pylorus in the stomach. Then you may increase the doses, giving them hourly or bi-hourly to begin with, and then double

and quadruple the quantity until the patient is having a fair amount of nourishment by the mouth.

Now a word or two about the causes of death in these cases. This is instructive from a general pathological, as well as from a surgical point of view. The causes may be put down roughly in this way: primarily shock and collapse, that is the great danger of all. It is easily understood when you consider the magnitude of the first accident, and the duration of the manipulations which are required to remedy the condition. Nothing could be more severe than the first lesion, and there is no abdominal section in which so much handling of the viscera is demanded as in these cases. The next cause is peritonitis. There is always some peritonitis in these cases, whether you like it or not, from the very start. We are rather prone to forget that this must occur more or less after the operation; if there were not we should not succeed; there must be adhesive inflammation. Where the boundary between the adhesive and non-adhesive process is we cannot say. But the inflammation must be within certain limits for recovery, and we are comforted by the feeling that it is often thus kept within bounds by the process of nature. Remember another thing which we are also prone to forget, that the peritoneal cavity has a most marvellous power of repair. If we treat it gently it can do more than we can if only we remove the greater part of the septic matter. I suppose we can never get the abdomen into an ideal aseptic condition, but if we remove the bulk of the irritating septic matters and do not damage the serous surface by rubbing or over-stimulation, this great lymph-sac will take care of the residuum, and also get rid of it. But we must not overstep the bounds of moderation, and damage the tissues which we are trying to help. Again, we may have not only a general peritonitis, but also a local peritonitis of a most severe form, which is capable of destroying the patient weeks after the original injury. This is that localised inflammation which leads to subphrenic abscess. The frequency with which this sequel occurs I cannot tell you. The records have been up to the present time deficient, but they will probably not be nearly so much so in the future when attention is directed to the necessity of careful observation. In my first case outside the hospital

the patient, who had recovered up to the tenth day, showed symptoms of localised peritonitis, and I felt sure there was a focus of suppuration somewhere, not in connection with the wound. On one occasion I actually went to the bedside with an anæsthetist prepared to operate for it, believing it to be in the subphrenic space on the left side. But I had not the courage to interfere further. I said, this patient has done fairly well so far, and after all there may be no collection of pus. But she died five days afterwards, and I found a huge subphrenic abscess, which I could have opened quite easily. In another case, my last, which was a success, a similar abscess formed on the right side. The wound was doing very nicely, the drains were acting well, and everything was satisfactory for many days. But ultimately the patient got a high temperature and a rigor, with pain on the right side. The symptoms of subphrenic abscess are, as you ought to know, very obscure. But I began to have a suspicion that there was an abscess, and as there was a little increase of liver dulness on the right side I put a needle through one of the lower intercostal spaces, and drew off 13 oz. of fluid. That settled the matter. The rest of the abdomen was quiet, and two days afterwards I cut in between the ribs and through the diaphragm and put in a drain between the liver and the latter. The patient recovered, and is now perfectly well, as I have heard quite recently, nearly a year after operation, having experienced no further trouble. This is a danger we must remember and try to forestall by cleansing the subphrenic spaces at first. But if subphrenic abscess occurs we must have the courage to operate on it, for many of such collections can be opened with success. Another trouble is liver abscess; that, too, follows in a good many cases. It is met with also in ulcer of the stomach without perforation, in consequence of pylophlebitis which has started in the veins of the stomach and travelled back into the portal veins. This is a very serious condition, and can probably only be operated upon with success in very chronic cases. Finally, there is bleeding from the ulcer when we reach it. In some of the cases, when the abdomen has been opened and the stomach has been manipulated a burst of blood has come from the ulcer which has necessitated most active measures. Some of these ulcers have been cauterised, others have

been treated by acupuncture, and others have been excised. I think if I were confronted with such an emergency as that while dealing with gastric ulcer I should excise the ulcer straight off and stitch in healthy tissue, and thus obviate the dangers of subsequent hæmorrhage. Another danger which has killed some of the patients who have been operated upon successfully is rupture of a second ulcer in another part of the stomach. That cannot be forestalled. If such can be seen, something may be done for it, but a certain proportion of such cases will always frustrate our best endeavours. Again, pneumonia has followed in some instances, as indeed after many abdominal operations. Probably in such cases the infection has taken place from the septic peritonitis. To illustrate this point I may say that in one of my patients it undoubtedly had spread through the diaphragm from the subphrenic abscess. It produced a septic pleurisy in the first instance, and then a septic pneumonia. This was present in that lung at the time of death, as proved by post-mortem examination. Of course pneumonia will occur in other ways.

The percentage of death from perforation of gastric ulcers, based upon the examination of large series of cases, has been variously stated. Amongst those who suffer from gastric ulcer, according to Habershon, who years ago went into the question, basing his study upon the post-mortem books of St. Bartholomew's Hospital, about 18 per cent. of patients suffering from gastric ulcer ultimately die of perforation. I am bound to say, however, that other observers have come to different conclusions. And there are those who hold that amongst patients suffering from gastric ulcer only about 13 per cent. die of all the complications. There are also those who put down the percentage very much lower. That I shall have to go into presently. In the meanwhile I may just say that on Saturday I went over 19 years of our hospital records and gathered up all the cases of gastric ulcer treated in the wards, and found exactly 200, only fifteen of whom died in the hospital at all, giving a total mortality of only $7\frac{1}{2}$ per cent.

(To be continued.)

DISCUSSION ON THE BENEFICIAL EFFECTS OF ONE DISEASE UPON ANOTHER,

At the North-West London Clinical Society, North-West London Hospital.

Dr. R. H. MILSON in the Chair.

(Continued from p. 20.)

DR. TURNBULL said he had under his care a child aged 4, suffering from whooping-cough. A very slight rash (chicken-pox) appeared, when the whooping-cough entirely stopped for the time being; on the rash subsiding the cough returned. In another case a child had pneumonia during an attack of measles. The rash disappeared, but the pneumonia ran the usual course, with a crisis on the fifth or sixth day, and shortly afterwards the measles eruption reappeared.

Dr. SIBLEY considered that the physiological causes of the influence of one disease upon another might be divided into two headings, both of a chemical nature—(1) the result of a toxin produced by one germ upon the development of another germ, and (2) the result of purely physiological chemical changes produced in the blood apart from germ disease. The latter could be subdivided again into various headings; for instance, uric acid. They were told by Haig that this chemical substance in the majority of people accumulated in the organs throughout life, and that when an illness occurred, either acute or chronic, owing to changes in the alkalinity of the blood, the uric acid became redissolved, and was got rid of by the kidneys and in other ways. It was due to that removal of uric acid that changes of temperament, &c., occurred in a large number of patients, as, for instance, in the case narrated by Dr. Campbell, the traumatism having probably determined the liberation of uric acid. Dr. Sibley narrated a few cases from his own observation. A woman was admitted to a hospital with scirrhus of the breast. She had been a subject of psoriasis for many years, over almost the whole of her body. The breast was amputated, and within a week every vestige of psoriasis had disappeared, although no internal or external remedies had been applied. Another case was one which came under

his notice some years ago of a lady who was addicted to alcohol who was practically drinking herself to death. She was seized in a drunken fit with cerebral hæmorrhage, and one side of her body was paralysed, which compelled her to keep her bed; but she lived ten or eleven years afterwards, and then succumbed to septic pneumonia following gangrene of the foot. He had seen in consultation an old-standing case of rheumatoid arthritis in a young woman who had been for many years an absolute cripple. A few months ago she had an attack of what seemed at first to be erysipelas, but which turned out to be erythema multiforme. She had high temperature which lasted a week or ten days, with great pain down the limb following the eruption. Since then the patient had gradually improved in all her symptoms, and they could not tell how far the improvement would go. Another case he related in which a gentleman had suffered a long time from asthma, but at sixty years of age he had pneumonia, and never again suffered from asthma, dying at the age of eighty from carcinoma.

The CHAIRMAN remarking upon Dr. Sibley's observations, said cases were known in which asthma came on early in life but subsided later, and others in which it did not occur until late in life; so that the case quoted might have been a coincidence. As to eczema, if a general practitioner saw a case of eczema in a child, he vaccinated it, which usually had a beneficial effect upon the eczema. He had not seen any effect produced upon whooping-cough by vaccination.

Dr. SUTHERLAND said it was a question whether a complication of pulmonary tuberculosis like pneumothorax or pleuritic effusion might be beneficial or the reverse. Some eight years ago he met with a case of pneumothorax which occurred in a patient who had been suffering from localised phthisis. He was showing marked constitutional effects, and during a fit of severe coughing pneumothorax ensued. The patient suffered severely for eight days, and at the end of that time Dr. Sutherland drew off a certain amount of air, as the symptoms of impending death were marked. He had to repeat the procedure three days afterwards, and three ounces of fluid came away with the air. From that time the patient improved, and made a very good recovery; the acute phthisical symptoms disappeared, he began to put on weight, and although there was a slight

amount of fluid in the chest, he left for the Cape four months after the attack. During the course of the following year he was aspirated once, but there had apparently been no return of the phthisis. He was at the Cape two years, and then returned to this country and was doing his work as an engineer at the present day. The point was what treatment they should adopt, and whether they should regard the effusion as beneficial in cases of active phthisis. Probably it was beneficial because it procured for the lung physiological rest, under which condition healing was very much easier. His own feeling was that under such circumstances the effusion should be left alone as long as possible,—that is, if the symptoms were not urgent.

Dr. CAMPBELL replied to the various speakers, and agreed with Dr. Sutherland that it seemed advisable to leave the effusion alone in a case of pneumothorax supervening upon pulmonary tuberculosis.

NOTES, ETC.

The Bacillus Mucosus-capsulatus (Bacillus of Friedlaender).—Since the first description by Friedlaender in 1883, and by Weichselbaum three years later, of an organism which was first called a coccus (pneumococcus of Friedlaender) and later regarded as a bacillus, occurring in acute fibrinous or croupous pneumonia, this bacillus has been found by many observers in various inflammatory processes in the lungs and other organs.

As specific characteristics of this organism were pointed out, its raised mucous-like growth on agar and blood serum, its nail-shaped growth on gelatin, its yellowish-grey growth with gas formation on potato, its failure to coagulate milk, absence of indol reaction, and decolorisation by Gram's method of staining; it was generally found to be constantly pathogenic for mice, variably so for guinea-pigs (fatal in about one half of the cases), rabbits and pigeons, and finally its polymorphism and capsule formation were regarded as quite specific. From time to time bacilli differing in some respects from the foregoing description have been met with in pneumonia and other affections, especially in inflammations of the middle ear and

upper air-passages, as well as in the peritoneum, liver, kidneys, and bladder, and lately also in the accessory sinuses of the nose. For these reasons there have often appeared in recent literature descriptions of a new bacillus pathogenic for man. Most of the authors have pointed out the striking similarity between these various organisms and the bacillus of Friedlaender. More or less striking differences in growth and other characteristics have nevertheless been assigned as sufficient reasons for establishing new classes.

The bacillus of Friedlaender, often called the pneumobacillus or capsule bacillus, is stated by Honl to occur in from 5·5 to 8 or 10 per cent. of the cases of croupous pneumonia, and more frequently, alone or with other bacteria, in bronchopneumonia. It is not uncommon in the usual complications of these affections. It has also been found alone, or with other bacteria, in inflammations in the liver, kidney, bile-passages, lachrymal gland, in the nose in rhinoscleroma, in ozæna, in the accessory sinuses in the nose, and in hæmorrhagic septicæmia of the new-born. It has also been demonstrated in the saliva of healthy mouths, in healthy noses, in the ground, dust, and air.

Fricke has been able to collect twenty-two bacilli from the literature which resemble the bacilli of Friedlaender. He points out the many identical features and includes them in one single group, with that of Friedlaender as the type. He proposes as a name for this group the *Bacillus mucosus-capsulatus*. He has found such organisms thirteen times, often mixed with pus cocci, in the tympanic cavities of thirty-two cases of otitis media, in one case in the antrum of Highmore, in ulcerative endocarditis, in the stools in seven cases of gastro-enteritis. He makes the differences in the pathogenic actions of these bacilli the basis for two groups. In the first group he places those bacilli that kill white mice after subcutaneous inoculation, and which have none or only a passive pathogenic action when inoculated into the abdominal cavity of guinea-pigs and rabbits. In group 2 are included those forms that are not pathogenic for mice on subcutaneous inoculation. Most of these were pathogenic for guinea-pigs.

Fricke insists that the so-called ozæna bacillus is identical with the *Bacillus pneumoniae* of Fried-

laender. Fraenkel, Paulsen, and Abel have all described a mucus-like encapsulated bacillus in connection with ozæna. Very recently W. T. Howard, jun.,* of Cleveland, Ohio, from whose article the above facts are abstracted, has briefly described ten cases in which he encountered the *Bacillus mucosus-capsulatus*. These cases varied very much as to the nature of the lesions. Four concerned purulent discharges from empyema of the antra or frontal sinuses; one was a case of puerperal septicæmia; another was a case of peritonitis and pyelitis after obstruction to urinary outflow from enlarged prostate. Another was a case of chronic peritonitis in a girl eight years of age, the peritoneal cavity containing a large amount of thick, creamy, tenacious pus, which on microscopic examination showed pleomorphic capsulated bacilli, and the culture showed a bacillus identical with the bacillus of Friedlaender. Another case was one of acute croupous pneumonia, with general febrino-purulent peritonitis. The bacilli found in these cases did not agree with each other in all respects, differing especially in the varying amounts of gas production, and varying in pathogenesis for guinea-pigs and rabbits. Howard concludes that this bacillus is much more commonly associated with acute and chronic infectious processes than is generally believed.

Joseph J. Curry † considers the *Bacillus capsulatus* with especial reference to its connection with acute lobar pneumonia. He found it as a pure or mixed infection in twelve cases of various kinds, including acute fibrinous pneumonia, acute endocarditis, otitis media, diphtheria, and tonsillitis. The connection of the *Bacillus capsulatus* with acute croupous pneumonia is especially interesting. The routine bacteriologic examinations of pneumonia as carried out in the Boston City Hospital, for instance, have shown that it is invariably due to the *Micrococcus lanceolatus*. One of the cases referred to by Curry throws considerable light on the cases of pneumonia which have been considered as due to the capsule bacillus in so far as it demonstrates that the growth of this bacillus may be and usually is so profuse as to obscure the growth of the diplococcus; examination of the sections from the lung in this case by the Gram

* 'Philad. Med. Journ.,' February 19th, 1898.

† 'The Journal of the Boston Society of Medical Sciences,' March, 1898.

stain, as well as with methylene blue, showed that the bacilli were, as would be expected, decolorised by Gram's method, while the diplococci were present in large numbers in the alveoli and in the pleural exudation; with the methylene blue stain the bacilli were found quite numerous, chiefly in the bronchi.

Journ. Amer. Med. Assoc., April 23rd, 1898.

"Ocular Thermometry."—On the 18th of January Professor Galezowski read before the Paris Academy of Medicine a paper entitled "*La Thermométrie oculaire*," and he publishes the substance of it in the '*Wiener klinische Rundschau*' for April 3rd. We all know now, he says, that the production of heat takes place in the depths of the tissues, as maintained by Richet, but that it is under the influence of the central innervation governing the activity of the heart and lungs. On making a close study of the complicated laws of animal heat, and endeavouring to make out their relation to the nutritive conditions of the eye, he came to recognise that the normal temperature of the eyeball, as well as that of its adnexa, varied with the age of the individual, and differed to a certain extent from the general temperature.

The author then mentions the well-known variations of temperature dependent on the age of the individual, the period of the day, the part subjected to thermometry, the waking or sleeping condition, &c., from a consideration of which he has been led to propose to himself the question of whether or not observations of the temperature of the eye might be of service in the diagnosis and treatment of deep-seated ocular disease. The question, which he satisfied himself had not been dealt with by the physiologists, resolved itself into three others:—(1) Whether or not it was practicable to measure the temperature of the normal eye; (2) what instrument would be best for such measurements; (3) whether or not any good would result from ocular thermometry.

By several years' observations he has been led to the conclusion that the eye will tolerate the contact of a suitable thermometer long enough for the temperature to be registered. With the aid of M. Peuchot, a Paris optician, he has contrived a thermometer, the "*ophthalmo thermometer*," which consists of a rectangular bulb slightly concave on

the side that is to be applied to the eye, and with the glass very thin, so as to heighten the sensitiveness of the instrument. The tube is bent in two places, so that the graduated portion stands upright when the instrument is in place.

As regards the third question, that of the utility of ocular thermometry, M. Galezowski admits that he cannot yet speak positively, for his experience thus far is not sufficiently rich. He feels confident, however, that his observations will eventually warrant him in stating that measurements of the temperature of the eye may be turned to good account in enabling us to note the development and progress of certain deep-seated ocular affections, such as chorioiditis, chorioid hæmorrhages, detachment of the retina, glaucoma, &c.

New York Med. Journ., April 23rd, 1898.

Streptococcus Pneumonia.—In an able article on "*The Clinical Course of Pneumonias in which there is an Infection with Streptococci*" (*'Boston Medical and Surgical Journal*,' April 14th) Dr. Francis P. Denny lays stress on the importance of a diagnosis of streptococcus pneumonia, whether with or without pneumococci, both for prognosis and for treatment. He says: "The family and friends always want to know when the crisis will come; and to be able to say in a case that there will be no crisis, but that the course will be a long and severe one, would certainly be of practical value to the physician. As regards life, the prognosis seems to be somewhat better than in the ordinary pneumonia." As regards expectant and symptomatic treatment at present in vogue, the diagnosis will not materially aid us; but "in the future, when we have an effective serum-therapy in pneumonia—an antipneumococcus and an antistreptococcus serum—the early differentiation of these two forms will be of the greatest importance."

The diagnosis must be made by sputum examination, and the principal clinical characteristics of streptococcus pneumonia are stated to be "the tendency of the local process to wander, the involvement of the upper lobe, the long and irregular type of the fever, and the much delayed resolution."

New York Med. Journ., April 23rd, 1898.

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* Specially reported for *The Clinical Journal*. Revised by the Author.

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LECTURES ON DEFORMITIES CONSEQUENT UPON INJURIES OF THE BONES AND THEIR TREATMENT.

Delivered at the City Orthopædic Hospital,
January 18th, 1898,

By JOHN POLAND, F.R.C.S.

LECTURE I.

[MR. POLAND first alluded to the frequency of these deformities as seen in orthopædic hospital practice, and to the almost complete absence of allusion to the subject in works on orthopædic surgery.]

The subject is a very large one, and comprises the deformities due to congenital fracture or other ante-natal lesion. I must in the first place exclude those cases which also come to us in great numbers, namely, those of deformity after excision of joints or other surgical procedure for disease. In many of these some further operation has to be performed to rectify the faulty position. [In dismissing this portion of the subject Mr. Poland showed skiagrams taken from a patient nine years after removal of an osseous wedge from the front of the knee for angular deformity following excision of the knee.]

As regards those of our profession in general practice, I do not believe that there is another subject which affects them so intimately as the deformity and impaired utility of the limbs the result of previous injury to bones or joints. This frequent and unsatisfactory result is a source of friction between the medical practitioner and patient, and unfortunately at times terminates in an action at law. The whole range of our subject is so vast that it is only possible for me to adequately deal with but a small portion of it this evening. Let me first deal with those deformities as they occur in infants and children, for they form the greater portion of the cases which come under our notice at orthopædic hospitals. In the

long bones of the extremities the deformities may be classified anatomically according to their existence in the shafts or at the epiphysal ends. The first deformities for us to consider are those due to intra-uterine fracture or injury to the long bones. The bone may be fractured *in utero* by a blow upon the mother's abdomen, by the convulsive movements of the *fœtus*, or by the entanglement of the limbs in the case of twins. The clavicle, humerus, radius, ulna, femur, and tibia are the bones usually involved. The deformities from intra-uterine fracture may be great from the displacement of the fragments, the fragments uniting in an angular position, or the fragments may remain ununited at or for several years after birth. Intra-uterine rickets appears from alteration in nutrition to predispose to fracture or bending of the bones. It is unnecessary for me to go further into this class due to disease, but wherever many of the bones are deformed from fracture at birth there is always a suspicion of congenital rickets. I must also discard from our consideration malformation due to other defects of ossification or development, or separation of the epiphyses in consequence of a syphilitic or inflammatory process, although in some instances it is difficult to say that the distortion after injuries is absolutely free from one or other condition existing at the time. Furthermore, an injury to the bone during intra-uterine life may not be so severe as to completely fracture the bone. A greenstick or partial fracture will not infrequently be discovered after birth, and union of the bone as a curvature in the shaft in a greater or less degree. I have lately had two well-marked instances under my observation—in one extending over two years (Fig. 1). In this latter case arrest of growth at the lower end of the tibia and curving of the bone occurred after intra-uterine fracture. At birth the tibia was half an inch shorter than its fellow, and at one year and nine months one inch. In the other case the skiagrams revealed no solution of continuity, but a general curving of the diaphysis. In other milder cases of curvature the deformity is due to contraction or spasmodic pressure of the uterus, as in the following case, which occurred lately in my practice at the Orthopædic Hospital. Both forearms and legs were much dwarfed, and on the outer aspect of these sections of the limbs there were dimples from cicatrices

over each of the bones about their middle as if from pressure. The cicatrix over each bone was adherent to it, and perfectly symmetrical on the two sides; in the case of the bones of the leg there appeared to be some displacement, especially with regard to the fibula. The forearms were curved anteriorly, and the feet were in the equino-varus position. The malposition in both extremities was easily remedied by the use of tin splints, and it only required in the case of the legs subsequent division of the tendo Achillis to correct the remaining equinus position for a perfect result

Fig. 1.

to ensue. The congenital deficiency or deformity of one or more of the toes is a remarkable coincidence in many cases of intra-uterine bendings and fractures of the long bones of the leg. My late colleague Mr. E. J. Chance described in his interesting and original work on 'Bodily Deformities,' a few instances of these deformities which had come under his observation. He thought very truly that the cicatrices which were present over the injured tibia, in one instance represented by a dimple over the projecting angle, were doubtless the result of a perforation of the skin by the sharp ends of the fragments.

The fibula seems to be deficient or absent in other instances, and I am of opinion that the dwarfing in these is really due to injury to the bone

during early intra-uterine life. An example of this sort came under my care at this hospital some few years ago; the tibia had been arrested in its growth, and was almost entirely absent, while the fibula on the outer side projected downwards below the foot. The latter was small and deficient in many respects, with only two toes. It projected directly inwards at right angles to the leg, but there were no depressed cicatrices (Fig. 2).

Fig. 2.

M. Blasius has reported a case of a healthy, well-formed child with an obtuse angular deformity at the junction of the lower and middle thirds of one leg. The skin presented a cicatricial-like retraction at the angle, where it was also unusually adherent to the bone. The ankle was very much drawn up, and the inner border of the foot directed upwards. The limb was smaller than the other, and had only two toes and two metatarsal bones in the foot. There was a doubtful history of a blow received upon the abdomen during pregnancy. Dr. W. J. Little as long ago as 1853 attributed these distortions to congenital atrophy of the leg and foot with malformation or contusion of the leg bones, giving rise to considerable curvature of the tibia forwards. In the examples he quotes there was no external malleolus perceptible when the foot was everted, the gastrocnemii and peronei muscles were contracted, and two or three toes were usually deficient, and at the most projecting part of the curvature of the bone a dimple-like depression of the skin existed, the integument being there more

adherent to the bone than elsewhere, reminding the observer of the cicatrix and prominence of the tibia after bad union of a fracture. This condition of the leg Dr. Little regarded as a malformation due similarly to over-distortions and to disorder of the muscular sensation producing inordinate muscular action. He gives a drawing of a case of contusion of the right tibia and fibula, from probably intra-uterine fracture of the leg bones with deficiency of the toes and valgus position of the foot.

A case of intra-uterine fracture of the tibia in the foetus has been recently described by Dr. Caviglia. The mother, twenty-six years of age, received in the fifth month of pregnancy a blow on the abdomen. The infant, a female, was normal except in the right lower extremity, the leg being bent at an angle, and the foot being in an equino-varus position. There was no trace of a fibula, and the tibia showed a fracture. Over the site of the fracture was a cutaneous cicatrix. The fifth toe was represented by a small node adhering to the metatarsus by a pedicle. The leg was somewhat smaller than the other. Caviglia thinks that there was an arrest in the development of the fibula, due to fracture of the tibia. Linear osteotomy and tenotomy were performed five days after birth, with improvement of the state of the limb.

Fractures of the femur, tibia, humerus, or forearm, produced at the birth by the expulsive force of the uterus or by the accoucheur in violent traction of the limbs, may, if not recognised at once, lead to subsequent serious deformity. These fractures have been especially studied by Küstner and others more recently, and need not engage our attention this evening. Epiphysial separation during childbirth I will also dismiss. In 1884 Dr. J. H. Brinton made some observations on intra-uterine fracture, summarising fifty-one cases already reported by different writers, and adding two cases of his own, and I must refer you to his paper for further opinion on the subject.

Now as to treatment. The angular deformity may be corrected by forcible straightening under an anæsthetic, and the limb put up in moulded pasteboard, or other light splint. In infancy after intra-uterine fracture the tendons must be divided before the bones can be replaced in position, as no doubt the muscles act upon the bone after fracture

and become contracted. This was well shown in the case of the congenital dwarfing of the legs and forearms and equinus position of the feet in the case I have already described. The varus position was corrected by malleable metal splints on the outside, as in the ordinary form of congenital equino-varus, and the tendo Achillis only required dividing for perfect restoration of the feet, although, of course, as I have said, these remain permanently shortened. Re-fracture should not be lightly undertaken in these instances of angular deformity, unless very great and increasing, on account of injuries to the epiphyses. Osteotomy may be required, as already mentioned, in some exceptional instances, but all these and other methods of treatment shall be considered in detail later on.

Taking next in order the *deformities which arise from fracture of the diaphysis in childhood*. Incomplete or greenstick fracture of the clavicle is perhaps one of the most common forms of fracture of the long bones which one sees in the out-patient department of a children's hospital. The mother usually brings the child with a history of its having had a fall a fortnight or so previously, that it had been examined and was said to have nothing the matter with it. A swelling is now suddenly noticed, and the child brought to the hospital. The callus from the fracture in this instance is often very large in amount, due no doubt to the child having been allowed to use its arm. With the arm fixed in a proper position the case usually does well. Similar instances of excessive callus around an undetected fracture are occasionally met with in other of the long bones of the extremities.

Deformities following fracture of the shafts of the humerus, bones of the forearm, femur, and bones of the leg in children are unhappily too frequent; and the great distortion from mal-position of fragments means want of skill or neglect in treating the original injury; but I shall deal with this later on. Excessive callus, too, is often combined with badly adjusted diaphyseal fractures in children, which of itself not only impairs the function of the limb, but causes serious pathological conditions by pressure on important vessels and nerves in its immediate vicinity. Paralysis of the median or musculo-spiral nerve is not an uncommon result of fracture

of shafts of the humerus in children, but in my experience interference with the function of the nerves by callus is not so common as some writers believe. The paralysis and consequent disability and distortion of the limb is in reality the result of injury to the nerves at the time of the fracture, or to their subsequent irritation or injury from displaced fragments. I have operated on many cases where the nerve has been directly pressed upon by a displaced fragment of the shaft of the bone, and has given rise to paralysis, &c., and in which removal of the displaced bony fragment has been followed by complete cure. The conditions due to the injured nerves are commonly overlooked at the time of the accident, and recognised only at a later date, generally at the time of the removal of the splints.

The treatment of the deformities resulting from all these extensive injuries to the shafts of the long bones I may also consider with similar deformities in adults.

Arrest of growth may follow a fracture of the shaft in children, and the importance of the direction of the canal for the nutrient artery and vein is of interest in this condition. Mr. T. Bryant mentions a case of arrest of growth of the humerus amounting to three and a half inches following fracture of the shaft at the age of eight years, which he attributed to injury to the nutrient artery. A precisely similar deficiency of growth and deformity came among my out-patients at the City Orthopædic Hospital in March, 1896. In this instance there had been a transverse fracture above the epiphysal line. The patient was a man twenty-two years of age, who had received a fracture twelve years previously. The end of the tibia was dwarfed in its growth and curved inwards, causing prominence of the internal malleolus, and the tibio-fibular joint slightly bent in this position, thus giving the foot a tilt inwards. The lower end of the fibula was considerably hypertrophied, and its subcutaneous surface marked by ridges. The foot was in a well-marked valgus position, in order to compensate for the inclination at the tibio-fibular joint, and the scaphoid came out prominently when the foot was on the ground. Other surgeons have recorded similar examples.

Non-union of the fracture of the shaft as we see it in orthopædic practice, usually the result of some small amount of violence, is again a not uncommon

occurrence. An analysis of sixty-three cases of ununited fracture occurring in the long bones of children was recorded by Mr. D'Arcy Power before the Royal Medical and Chirurgical Society in December, 1891. He believed that until the publication of the very valuable paper on this subject by Sir James Paget in his 'Studies of Old Case-books,' the occurrence of non-union in children had been almost wholly neglected. The conclusions arrived at by Sir James Paget are entirely borne out by the cases collected by Mr. Power.

Juxta-epiphysial sprains.—Although no deformity is present at the time of the injury to the long bone, it may subsequently result from arrest of growth due to osteitis, to epiphysial sprain, or to contusion. In infancy the injury of the juxta-epiphysial region may be of the character so well described by Ollier, and may be considered at this time as a simple sprain, and yet be followed by arrest of growth. He points out that the more active the proliferation of a tissue the more susceptible is it to morbid changes at any particular time, and that this is particularly true of the growing bones. Consequently in infancy the effects of injuries to the rapidly developing bones are especially to be feared.

We pass now to another very large class of deformities, due to traumatic separation of the epiphysis at or near the joint of the limbs. They are numerous and commonly characteristic of the original lesion of the diaphysial end of the bone. In some, however, the deformity and loss of function of the joint or limb is quite unlike the injury which took place in the first instance. Moreover, if the injury has been overlooked, and the child allowed to use its limb, the epiphysis may have become gradually displaced, and lead to serious after consequences. In others the precise condition of the displaced fragments is often most obscure, often complicated with dislocation and rotation of a part of the epiphysial fragment, and the joint often hampered in its movement by exuberant callus or completely fixed by displacement of some articular portion. Through our present imperfect knowledge of their pathological anatomy it is usually quite impossible to make an exact diagnosis if many weeks have elapsed from the time of the accident. The Röntgen process will undoubtedly help us much in elucidating many

of these injuries, for specimens are seldom to be found in pathological museums.

Deformity following separation of the epiphysis.—The modelling process after union has occurred is happily effected so rapidly and completely, even in many instances in which the reduction has not been quite perfect, that within a few months after the injury it is almost impossible to detect the precise situation of it. A more or less considerable enlargement of the diaphysial end, as compared to the sound side, is always present, and is especially noticeable when the periosteum has been detached to any extent. This increase in volume of the injured bone remains for a year or two, but disappears more rapidly the earlier the age of the patient at the time of the accident. Deformities from shortening have hitherto been unfortunately too frequent. The displacement may be so great that the vessels of the diaphysis being no longer in contact with the epiphysial cartilage, the nutrition and function of the latter may be seriously interfered with. It should, however, only be a temporary derangement until the subsequent absorption of callus, which rapidly unites the separate ends as a rule. The surface of the diaphysis still in contact with the epiphysis is quite sufficient to carry on the vascular supply in a very short period of time. Permanent deformities with vicious union and impaired movements of the joint from displaced fragments are usually the results of imperfect reduction; it may be from want of care in reduction or proper appreciation of the exact form of the lesion, or from the injury having been mistaken for a dislocation, and an attempt made at reduction and no splint applied. Many of these old deformities persisting after separation have been mistaken for neglected dislocations, and attempts made to reduce them. Certainly in some instances the diagnosis between the two, after some interval of time, is somewhat difficult.

Ankylosis and impairment of the neighbouring joint after separation of the epiphyses and long bones are of common occurrence. The joint may be left stiff or even deformed as a result of inflammatory extension from the epiphysial injury in its neighbourhood, and Foucher has correctly described intra-articular separations as having the same prognosis as a true intra-articular fracture. In epiphysial separations about the elbow-joint incomplete recovery of the function of the limb is

especially common. In cases of permanent stiffness of a joint following an injury in which some irregularity of bone is to be felt about the conjugal line, it is highly probable that the original injury has been an epiphysial detachment, and not a dislocation.

Ankylosis due to arthritic inflammation may be occasioned not only by the injury to the joint at the time of the accident, but also by displaced fragments improperly reduced keeping up the inflammatory process. These displaced fragments also lead to the production of much irregular and vicious callus. Exuberant callus from incomplete reduction of epiphysial injuries in many situations, as at the lower end of the humerus, also limits the movements of the joint very extensively.

Paralysis of nerve due to inclusion by callus has been noted in a few instances at the lower end of the humerus after epiphysial separation.

Non-union.—No authenticated case of non-union of the separated entire end of a long bone has been recorded, and no museum specimen of it is in existence. Should non-union occur after such an injury, I am strongly of opinion that it will be found to be due to imperfect fixation, or from the injury being overlooked and treated as a sprain, and mobility of the fragments permitted.

Arrest of growth of the long bones after separation of the epiphysis.—A proper study of interference with the growth of the long bones commenced about the middle of this century, but so long ago as 1632 a Neapolitan surgeon, Severinus, drew attention to the separation of the upper and lower epiphyses of the tibia as well as to the deformities of the knee and foot which result from such separation. Roqnetta in 1834 clearly described shortening of the arm, deflexion of the hand, displacement of the foot and knee, and shortening of the leg as being most often the result of an undetected epiphysial separation; but this surgeon wrongly attributed congenital dislocation of the hip to epiphysial separation. It is undoubtedly true that separations are much less frequent during the first ten years of life than during the second decade, and the separations more likely to be followed by arrest of growth in the former than in the latter, when the bone is more developed and less under the influence of the epiphysial cartilage. Arrest of growth occurs most often at the upper

epiphysis of the humerus, at the lower epiphysis of the femur, tibia, and radius, by reason of the late period of their union with the diaphysis, and their being the most frequent seats of separation. Ollier is probably correct when he states that the reason why epiphysial separations are so seldom followed by arrest of growth is due to the fact that the injury seldom occurs in the cartilage itself, but rather in the adjoining ossifying spongy tissue, or in the juxta-epiphysial region immediately adjoining.

Deformity consequent upon arrest of growth.—The diminution in length of a long bone from diminished growth means loss of symmetry, and in many instances loss of the proper function of the injured limb. These deformities have been carefully studied by M. Poncet, especially in the parallel bones of the forearm and leg, as well as by Vogt and some other German authors. When the arrest of growth involves one of the two parallel bones of the forearm or leg, deformities which are progressive up to the time of the union of the epiphyses of the neighbouring bone are often seen, and are of great interest. The retardation of growth in the one bone, with pressure from the continuity of growth in the other, leads to considerable deviations in the contour of the articular surfaces and the peripheral parts of the limb. For instance, when the radius is involved, the ulna continues to grow, obtaining its fulcrum from the humerus, drives the hand outwards, and produces a valgus position of the hand. The opposite is the case when the ulna is injured.

Injuries to the epiphysis are frequently followed by inflammation passing on to suppuration, periostitis, and often osteomyelitis and necrosis, and subsequent deformity through arrest of growth is not uncommon. Indeed, these inflammatory complications are the most frequent causes of arrest of growth after epiphysial injuries.

I will now take in order the deformities as they occur after separation of the epiphyses of the long bones in the upper and lower extremity.

Deformity and shortening from arrest of growth after separation of the epiphysis of the clavicle is a possible though unlikely occurrence after this injury. Mr. Maunder draws too gloomy a picture of the consequences of arrest of growth of the clavicle when he says that "if it ceases to grow while the rest of the body is still undeveloped, its

good offices towards the corresponding extremity and half the chest fail, and with its failure I should fear an enfeebled extremity, a curved spine, diminished chest capacity, partially developed lung, ill-aërated blood, feeble circulation, an ill-nourished body generally—a list of things likely to engender serious disease hereafter.” The recorded instances of non-union after fracture of the clavicle at the inner end do not, however, bear out Mr. Maunder’s opinion.

Deformity after separation of the upper epiphysis of the humerus due to incomplete reduction of the fragments is very characteristic, and is also seen in certain fractures of the surgical neck, and described since 1645 by Boyer, Decamps, and many others. There is slight flattening of the shoulder with some atrophy of the deltoid, biceps, and muscles of the arm, half to one inch shortening of the long axis, the arm directed backwards and outwards, and the forearm semi-flexed, marked impediment to the movements of the arm, although the hand can be placed on the opposite shoulder, and slight depression below the arch from stretching of the deltoid. The head of the humerus is in position, and follows the movements of the arm, showing that the fragments are well united. In front of the shoulder, one and a half to two inches or more below the acromion process, there exists a very considerable and characteristic projection, somewhat rounded in front but flattened above. This projection of the diaphysial end forwards and inwards towards the coracoid process may be so great as to be in contact with this process; or, in some exceptional instances, there may be only some irregularity and thickening about the epiphysial line. There is often considerable loss of function, loss of active power in the hand and whole extremity, while passive movements, although free, are limited. The deformity is so very much like dislocation that attempts have frequently been made to reduce the supposed displaced head of the bone.

Arrest of growth of the humerus after epiphysial separation is not infrequently met with, for the reason that the upper epiphysis of the humerus is mainly concerned in the growth in length of the bone. Arrest of growth of this bone may also follow fractures other than epiphysial separation. In the museum of Guy’s Hospital there is a specimen of arrest of growth of the left humerus for three and a

half inches in what was probably a fracture of the shaft at eight years of age. I removed it, as well as the opposite humerus, while acting as dresser to Mr. Bryant many years ago.

Ankylosis of the elbow and deformity, the result of displacement backward of the lower epiphysis of the humerus, is unfortunately only too common, while a transverse fracture of the lower end of the humerus immediately above the epiphysial line displaced forwards is indistinguishable from pure separation of the epiphysis with the same displacement. A marked example of the latter injury came under my notice in 1893, where I was obliged to excise the joint for the injury, on account of the great deformity and loss of function. A successful result followed. Besides the fracture of the humerus in this case immediately above the epiphysis, (so that the track on each side almost touched the cartilaginous epicondyles, in the centre it was rather more than a quarter of an inch above the epiphysial line), the cartilaginous epiphysis of the olecranon was also separated, and displaced rather more than half an inch backwards off the upper end of the ulna. I have also seen great deformity accompanied by hyper-extension at the elbow-joint in old separation with displacement backwards. This class of deformity was well exemplified in another case which came under my care in 1895, but time will not allow me to discuss this in its peculiar features. Displacement outwards of the epiphysis, either as a simple separation or combined with fracture of the diaphysis, which is often an oblique and extensive one, with a consequent deformity, is not uncommon; while ankylosis of the elbow after either of the injuries just enumerated, although frequent, is not more common than after injury in the neighbourhood of other joints, although it is possible that the injuries may be followed by a partial arrest of development or irregular growth in the lower end of the humerus. The arrest of growth in the length of the arm after these injuries is scarcely worth consideration from the small degree of increase in the length of the humerus which takes place at the lower end of the bone. Paralysis from implication of nerves in separation of the lower epiphysis of the humerus may occur, namely, to the musculo-spiral, median, and ulnar, which are each of them liable to be involved in the abundant callus so often present, and which extends up the humerus for some dis-

tance. Their injury may give rise to paralysis, loss of sensation, hyperæsthesia, &c. Resection of the callus pressing upon the nerve may be required, so that the nerve may be set free. I have already alluded to the fact that the nerves are more commonly injured at the time of the injury, and that the paralysis is often overlooked. However, paralysis of the musculo-spiral nerve produces the characteristic and familiar wrist-drop, which is little likely to be overlooked. The existence of nerve lesion remaining after the examination of the power, sensation, and attitude of the limb, can generally be set at rest by testing the electrical condition of the muscles and nerves by the faradic or galvanic currents.

Deformity of the elbow following separation of the internal condyle of the humerus has come under my observation in several instances, but it is never very great. According to some surgeons no deformity should be expected, although Gross says that few cases recover without a certain degree of deformity or even ankylosis. But displacement of the epicondyle is usually downwards and forwards, while there may be some atrophy of the muscles connected with the epicondyle, or some paralysis may be present, due to injury of the ulnar nerve. In many instances a dislocation of the elbow-joint has been present and the simultaneous detachment of the epicondyle unfortunately overlooked. Deformity after condylo-trochlear fracture in children, with displacement upwards of the fragment, produces cubitus varus. This is known in America as the gunstock deformity, the normal humero-ulnar or carrying angle becoming reversed. The deformity is often very great where this form of fracture is complicated with dislocation. Osseous ankylosis, exuberant callus, paralysis or neuralgia of the ulnar nerve are at times met with, while arrest of growth will be very slight.

Deformity after condylo-capitellar fracture is perhaps one of the most common forms met with at the elbow. A marked example of this came under my care at this institution in 1895, the fractured portion of bone consisting of the capitellar portion of the lower epiphysis and a portion of the outer part of the diaphysis still connected with the epiphysal piece was displaced upwards and outwards, and remained ununited in this position (Fig. 3). It was the case of a boy æt. 9 years, who slipped and fallen upon his left elbow. He

had been treated by plaster-of-Paris bandages for six weeks, when passive motion was commenced. When he came under my observation he could use his arm freely, but it was weaker than the other; flexion, extension, pronation, and supination were good. From the front as well as the back the elbow looked widened. On the outer side there was a large fragment of the outer portion of the lower end of the humerus, freely

Fig. 3.

moveable upon the shaft. With this fragment the radius was still connected, and on its head rotated. The distance between the epicondyles behind was one and a quarter inches more than on the uninjured side. During flexion the olecranon was in the same plane as the epicondyles, having passed upwards and outwards between the two fragments and the lower end of the humerus, so that when the elbow was placed in this position on the table each of these osseous projections touched the surface of the table. The skiagram showed clearly the position of the bones, as well as

some dark spots which represented calcareous nodules in the subcutaneous tissue of the arm and forearm. The Royal College of Surgeons' Museum contains an excellent specimen of the articular ends of the bones forming the elbow-joints removed by excision. In this example the separated epiphysis of the capitellum and external condyle in one piece had been displaced outwards, and united to the shaft in that position.

There is also in the Guy's Hospital Museum a specimen showing a portion of bone removed from the elbow-joint by excision by Mr. Bryant. On careful dissecting out the parts and removing the fibrous tissue which united the ends of the bone I was able to make out the precise condition of the fragments as follows. There was partial dislocation inwards of the radius and of the ulna, with separation and outward rotation of the capitellar portion of the epiphysis. Combined with these injuries there was detachment of a portion of the internal epicondyle. The complete fixation of the elbow-joint in this instance by the displacement of the epiphysal fragment rendered excision of the joint absolutely necessary.

A well-marked specimen of displacement of the capitellar, and external and epicondylar epiphysis of the left humerus, with a fracture of a small part of the diaphysis united, from a lad eighteen years of age, I shall now show you. It was from a skeleton supplied to me many years ago, and I need not detail to you the probable deformities and loss of function which had evidently been present in this case.

Ankylosis from the two injuries which I have just described, and which are intra-articular, must always result. The ankylosis, although often temporary, may be permanent, and does not arise so much from arthritis as from the intervention of the separated fragment or improper reduction of it, giving rise to alteration of the normal articular surfaces. In other instances the olecranon fossa is so filled up with inflammatory osseous material as to greatly hamper or entirely prevent the movements of the joint.

The deformities of cubitus valgus and cubitus varus following fracture into the elbow-joint were then described in detail. [Mr. Poland proposed in his next lecture to continue the consideration of the deformities after epiphysal injuries of the remaining part of the upper and lower extremities.]

MEETING OF THE SOCIETY OF ANÆSTHETISTS.

At 20, Hanover Square, March 17th, 1898,

The President, Dr. DUDLEY BUXTON,
in the Chair.

Mr. McCardie read the following communication upon three clinical cases.

Death during the administration of an anæsthetic at the General Hospital, Birmingham. On December 15th last year M. R—, æt. 5 years, an in-patient, and a delicate rather pale-looking child, was brought to the theatre for removal of both tonsils and adenoid growths of the naso-pharynx. The tonsils were stated to be of unusual size, and the statement was verified. There was no marked difficulty in breathing. The lungs and heart were examined and nothing abnormal was found, nor was there any history of recent cough. Afterwards we heard that about six months previously the child had been treated in the casualty department for a slight attack of pleurisy.

The patient being placed on the table in a perfectly level position, the A.C.E. mixture was administered on a Skinner's mask, and without any trouble the child was soon anæsthetised. Then she was taken to the operating room and placed for operation, so that the head was level with the rest of the body. From this position she was not moved, nor was the head raised or depressed until attempts at resuscitation were made. The temperature of the theatre was 65°, and the child was warmly clad. As the patient was lightly under and moved occasionally, the A.C.E. mixture was continued on a double fold of flannelette. A gag was inserted, and one tonsil removed. After the removal of the first tonsil chloroform was given in the same method. When the second tonsil was removed there was fairly free bleeding, but all through the pharynx had been kept free from blood, and none entered the larynx.

Up to this time the breathing, though light, had been regular and unembarrassed, the pulse regular and rather small, and the colour not much paler than before. The child, after removal of the tonsils, began to move and recover conjunctival reflex, so chloroform was given as before pre-

paratory to removal of the adenoids. Hardly had she taken two or three light breaths than the pallor became deathly, the eyeballs fixed, the pupils dilated, the pulse was found to have disappeared, respiration stopped, and after that the only suspicion of life excited was by the occurrence of two gasps at an interval of a minute or so during artificial respiration. The usual means of resuscitation were at once employed, and continued for about half an hour. Inversion, artificial respiration, with pulling out of tongue, stimulation of heat and cold to the chest, strychnine injections, &c., but nothing availed, the only response being the two gasps above mentioned.

Air entered freely through the glottis into the chest during artificial respiration. Two drachms A.C.E. mixture, and about one drachm of chloroform (of which by far the greater part was wasted because the flannelette was not continuously applied) were used.

The whole time occupied would be about five to seven minutes in producing anæsthesia, taking out tonsils, and swabbing out the mouth.

At the post-mortem examination by Dr. Powell White, pathologist to the hospital, practically nothing abnormal was found. There was one slight adhesion of one lung, and a very little blood in the trachea. The right side of the heart, especially the right ventricle, was dilated, and the blood was fluid.

In his (Mr. McCardie's) opinion death was due primarily to syncope, secondarily to shock and the anæsthetic. The child was delicate and of a nervous temperament, and would be more liable to shock than most children; the tonsils were unusually large and difficult to excise, and the blood lost meant more to the patient than it would to most children; so that the effect of shock, added to the dilating and depressing effect of the chloroform given in the mixture and separately on the vascular system and heart muscle, immediately disposed to that factor of syncope which completed the disaster. As regards posture, the horizontal one was throughout maintained.

By the kindness of Mr. Gilbert Barling, surgeon to the General Hospital, Birmingham, the following case of death under chloroform was reported:—Miss R—, æt. 63, was admitted under care of Mr. Barling on December 30th, 1897,

with tumour of the bladder. It was found that she also had glycosuria, but there was no excessive thirst or wasting, nor was there excess of urine. On January 7th examination was commenced without an anæsthetic, but it was so painful that it was decided to administer chloroform, for which the patient had been prepared in the usual manner. Chloroform was administered on a piece of lint by the house surgeon of the case, and the patient took the anæsthetic very well, without struggling or cyanosis, and the pulse remained of a good volume and of ordinary frequency. Having completed his examination with the cystoscope, Mr. Barling washed his hands, and was just going to take the anæsthetic to continue it, that the house surgeon might also examine the patient, when suddenly the breathing stopped. Up to this time the patient had been breathing well, the pulse was good, and the pupil was moderately contracted. When the breathing ceased the pulse was still beating, and was felt for certainly half a minute later. Directly cessation of breathing was noticed the tongue was well drawn out of the mouth with forceps and the mouth kept open with a gag; the head was pulled over the edge of the bed, and artificial respiration begun, which was continued for over an hour. Hot flannels were applied over the cardiac area, strychnine and ether were injected subcutaneously, and finally six ounces of blood were taken from the left external jugular vein. Air entered the chest freely, but there was not the faintest attempt at natural breathing.

Post-mortem examination made by Dr. Powell White, pathologist to the hospital, showed slight fatty degeneration of the heart muscle and atheromatous changes of the aortic and mitral valves, but there were no vegetations on the valves, which appeared competent; there were no distinct changes found in the lungs, kidneys, or brain.

Mr. F. Marsh, surgeon to the Queen's Hospital, Birmingham, courteously allowed the reading of the following record of a death during the administration of chloroform.

T. V—, æt. 39, who had formerly been a cab driver, was admitted an in-patient of the Queen's Hospital on August 21st under Mr. Marsh. He was then found to be suffering from a pyonephrosis which had burst into the perinephral tissues, and was pointing in the left lumbar region.

A simple incision was made in this region, and

over a pint of pus was let out. The anæsthetic used on this occasion was the A.C.E. mixture, and the patient bore the administration without showing any unfavorable symptoms, though the following day he had a slight attack of bronchitis. He made a rapid recovery after the operation, and was discharged in November with a sinus leading down to the kidney, through which urine constantly trickled. This became so irksome to him that by his own request he was readmitted in the second week of December, with the idea of having a further operation performed. On December 31st it was decided to explore the kidney and see if it were possible to close the sinus. The anæsthetic chosen on this occasion was chloroform, the grounds for this choice being—in the first place his kidney mischief, which probably affected both organs, contra-indicated ether; secondly, his previous attacks of bronchitis pointed to the possible danger of ether; thirdly, he was evidently suffering from a degenerated heart, which possibly would not tolerate the excitation and strain of ether administration.

The chloroform was given by the open method, *i. e.* on an Esmarch's mask and a single layer of lint, and it was given in small quantities at a time. The patient took the anæsthetic well at first, though he was rather a long time going under, and towards the end struggled a little. He then suddenly lost his conjunctival reflex, and began to breathe deeply. The anæsthetic was stopped at this point and the patient was rolled over on to his right side preparatory to making the lumbar incision. The breathing now became worse, being shallow and irregular; the face was cyanosed and the pulse became feeble. He was immediately turned on to his back again, artificial respiration was commenced, and 30 minims of ether were injected hypodermically and followed by 5 minims of strychnine. But in spite of these efforts his respiration and pulse failed (apparently together), and though the artificial respiration and heart percussion, &c., were kept up for forty minutes, he showed no signs of returning to life. On post-mortem examination the left kidney was found to be completely disorganised, and the right to a large extent. The heart walls were extremely thin, and showed a considerable amount of fatty infiltration. There were signs of old tubercle at both apices. The bladder wall was enormously thickened, and the ureters were dilated.

Mr. McCardie said he should much like to know whether in cases of heart failure any members of the Society had tried the method of percussion of the heart, for he had heard it well spoken of once or twice, and in one case mentioned, the result was simply marvellous, the method having been successful after artificial respiration and inversion for some little time had failed. The interval between the apparent death and commencement of percussion was judged to be about three to four minutes.

Again, in the kidney case Mr. McCardie said he would be glad of expressions of opinion as to the supposed deleterious effect of ether on the kidney.

The PRESIDENT said these cases were of considerable interest. With regard to the first case one might ask the question which they often did ask themselves when reading the reports of the death of children who had been operated upon for removal of tonsils and post-nasal adenoid growths, namely, how long the child had been kept without food. There could be no question that in short and trifling operations in which a comparatively large quantity of blood was lost, it was most important that the patients should not be kept too long without food. It had occurred to him many times that the rule about food which forbade the patient having food for perhaps many hours had frequently led anæsthetists and surgeons into error. A case was recorded at a throat hospital in London where a child certainly died from syncope due to extreme exhaustion. The child had been kept the whole morning without food, and the operation was eventually performed late in the afternoon. He had no doubt it died out of hand through vital exhaustion due to lack of food and loss of blood. He would, therefore, like to hear from Mr. McCardie whether in the first case he had described, the patient had been kept unduly long without food. Mr. McCardie had mentioned in his remarks that the quantity of blood lost, although not large, was more than the patient could stand. As to the third case, they were told that chloroform was given rather than some other anæsthetic—(1) because of advanced kidney disease; (2) because there was a tendency to bronchitis; and (3) because the arteries were degenerated. That, of course, was quite consonant with the rules laid down in the text-books.

Whether those rules would not have to undergo revision he thought was a very open question. Certainly it had never been shown that ether properly given was very liable to produce kidney trouble, nor was more likely to give rise to extensive, bad sequelæ *quâ* the kidney than were other anæsthetics. No doubt it was true that in very prolonged operations upon the kidneys, or upon patients in whom the kidneys were diseased, a large quantity of ether might produce a great deal of irritation, and pre-existing albuminuria might be increased. One knew, however, that in patients without pre-existing albuminuria chloroform produced that condition more than did ether. On the other hand, one had to remember that the quantity of chloroform taken was very much less than the quantity of ether inhaled, and he imagined that the irritation of the epithelial cells of the kidney was directly in proportion to the amount of the anæsthetic which had to be eliminated. With regard to bronchitis, it had been asserted very strongly by a French surgeon—M. Lépine—that he noticed that where absolutely pure ether was given bronchitis did not ensue; and many of them knew that cases of bronchitis and nephritis in this connection were due to the inordinate and faulty way in which ether was very often given. In America the plan too often adopted consisted in pouring the ether in very large quantity upon a cone, and holding the patient down until he was saturated with ether. Under those circumstances nobody would question for a moment that bronchitis or nephritis might arise. But given carefully by a skilled anæsthetist it was very questionable whether ether did give rise *de novo* to bronchitis or kidney trouble. Probably the wet towels spread over the patient's body, and the exposure of large cutaneous surfaces, necessitated by the surgical toilet, had in many cases produced bronchitis which was attributed only to the ether. With regard to the degeneration of arteries, if one took the trouble to make tracings of the alteration of the size of the artery under ether, one found that the stimulating effect of ether was comparatively trifling, and occurred more in the early stage, namely, that of excitement, which could be entirely abrogated by the use of nitrous oxide gas. It appeared probable that if gas and ether had been given in the third case related by Mr. McCardie, the patient might still have been alive.

Mr. A. CHAPMAN said in reference to the remarks about ether and bronchitis he had recently had two cases where an anæsthetic had to be given during an acute attack of bronchitis. In the first case he had previously, some months before, given chloroform to an old man aged sixty-seven, and had a great fright with him because he had a distinct attack of syncope on the table, and the operation had to be completed under ether. The second time he had to give it was following an accident, in which the patient had to have amputation above the ankle performed. He gave ether in that case at the outset, and instead of the bronchitis being worse after the operation it was rather better. In another case, also an old man who had an attack of acute bronchitis, who had to be operated on for strangulated hernia, he administered ether, and found no ill effects from it.

Mr. TYRRELL said that long ago a patient of Mr. Allingham, sen., asked him (Mr. Allingham) whether he ought to have ether as an anæsthetic, as he had asthma. Mr. Allingham replied that he also had asthma, and he took ether to do him good. Probably it brought up the expectoration and produced relief in that way. He thought the remark interesting in the present connection.

Mr. CARTER BRAINE said he would like to ask, in regard to the first case, whether the head was dependent over the edge of the table or whether it was raised. He noticed that the A.C.E. mixture was administered up to the time the operation commenced, that the anæsthesia was not sufficiently long for the operation to be completed, and therefore chloroform was substituted on two folds of flannel. He did not consider this method of giving chloroform upon lint after the patient had been anæsthetised with A.C.E. desirable in throat operations. He had himself never had an adenoid case in which it was necessary to go on with chloroform after A.C.E., but should he meet with one he would use Junker's inhaler, considering it far safer under the circumstances. He felt bound to say that in those cases where tonsils and adenoids had to be removed he preferred, whenever possible, to have one of the tonsils removed a day or two prior to the major operation, as it considerably reduced the risk run. He noticed that Mr. McCardie said there was a little blood in the trachea, and he would like to hear whether Mr.

McCardie had any explanation to offer as to how it got there—whether it was during the operation or afterwards. With regard to the second and third cases, both chloroform cases, he would not have had the slightest hesitation in administering ether in either of them. He had had a great experience at St. Peter's Hospital for Stone in such cases, and almost invariably administered nitrous oxide and ether, chloroform being used in not more than 5 per cent. of the cases. He would like to ask whether the bladder was being distended in the second case, as he had noticed that when the bladder was being much distended there occurred deeper respirations, and consequently a greater intake of the anæsthetic.

Mr. McCARDIE, in reply, said that the child whose case he first related had a light meal of bread and butter and tea at six o'clock in the morning and that the operation was begun at 10.30, according to the usual rules at the hospital. In the third case, the President spoke of giving chloroform in cases of advanced kidney disease. He quite agreed that in ordinary cases, unless the operation was very much prolonged, ether did not cause any serious after-symptoms. He had no opinion to offer regarding the production or increase of albuminuria by chloroform or ether. He was very glad to hear of the French surgeon's statement that pure ether did not cause bronchitis. He had always used the "pure methylated ether." He did not know whether ether prepared from rectified or methylated spirit was used in most London hospitals. Sometimes patients had some bronchitis and other pulmonary ill effects from the methylated ether, but he did not think they would be able to trace anything more serious than after the use of the purest ether. He would like to ask Mr. Chapman whether, in the case whose bronchitis was better after the administration of ether, how long it was administered, and whether it was given by the open or closed method. As to the position of the patient in the first case, the head and body were perfectly flat until artificial respiration was begun, and then the head was depressed. He did not agree with the speaker who thought it was unwise to administer chloroform after A.C.E. He always preferred to begin with a mixture containing ether first, and then afterwards, if necessary, to continue with chloroform, rather than begin with chloroform. As to the removal of one tonsil

before the major operation was begun, *i. e.* removal of adenoids, he thought that was purely a matter for the surgeon to decide. As to how the blood got into the trachea in the first case, there was no question during life or during the performance of artificial respiration of any undue amount of blood being in the trachea or larynx, which was kept perfectly free by frequent swabbing. The blood probably oozed in after death. Regarding the question of distension of the bladder in the second case he could not speak from personal knowledge; he had read the reports as they were furnished to him, but he was told that the bladder had not been distended at all, and that there was no operation, merely examination. Neither could he answer the question as to cyanosis in the third case, or speak as to the length of time taken to anæsthetise the patient.

Mr. CHAPMAN, in reply to the question of Mr. McCardie, said he used methylated ether, and it was given by Clover's inhaler.

(To be continued.)

The X Ray in Fractures.—Dr. George W. Crary said that he had been struck with the fact that a well-marked fracture might be present without showing in the X ray at all. A Röntgen picture was presented from a case of Colles's fracture, in which the fracture could not be discovered even in the negative. Four surgeons had examined the case, and all had obtained easily the most definite evidence of such fracture. Where there was no displacement, and the fractured ends were in contact, the picture might not show a fracture at all. This fact was of some medico-legal importance. Another fact that had impressed him was that the callus would not always be shown in an X ray photograph. Röntgen pictures were presented from a case of non-union, which had been brought to him two months after the injury. He had succeeded in getting union, but had been unable to reduce the deformity. It had been a case of fracture of both bones of the forearm. Four months after the receipt of the injury, while there had been distinct and firm bony union, the Röntgen picture had appeared to indicate that union had not taken place. Apparently these pictures would not prove that union had occurred unless the callus was old.

New York Med. Journ., May 7th, 1898.

THE SURGICAL AFFECTIONS OF THE STOMACH, AND THEIR TREATMENT.

A Course of Lectures delivered at University College during February, 1898, by

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No. V.

GENTLEMEN,—We have now to consider the second division (*b*) of GROUP I, the Emergency Operations, namely, that for hæmorrhage. The latter is believed by many observers to be a cause of death in about 5 per cent. of those suffering from gastric ulcer.

5 per cent. Müller makes it higher, namely, 11·6 per cent., and Steiner gives 6·3 per cent.; Debove and Rémond 5 per cent. Other observers give figures which are close to these last, so I think you may take it that of those afflicted with gastric ulcer about from 5 to 6 per cent. die ultimately of hæmorrhage. Of course no operation would be undertaken by any surgeon in his senses for the excision or other operative treatment of an ulcer simply because the patient had some bleeding from the stomach, that is if it were a case of slight hæmatemesis or melæna. This greatly limits the number of operations performed for bleeding. But where a patient has had one or two serious attacks of profuse hæmorrhage, and is obviously dying of the loss of blood from perforation of a large vessel, operation is justifiable to attempt the arrest of the bleeding, unless of course the individual is too weak to bear any operation. So far very few such procedures have been carried out successfully. A

Table XX (various authors).

Percentages of Causes of Death from Gastric Ulcer.

	Debove and Rémond.	Gerhardt.	Welch.	Habershon.	Müller.	Steiner.	Hauser.
Perforation	13·0	13·0	6 to 5	18·0			
Hæmorrhage.....	5·0	3 to 5	3 to 5	11·6	6·3	
Pyloric stenosis...		10					
Inanition	5·0						
Pylephlebitis.....			4 to 5				
Cancer							5 to 6
Tuberculosis.....	20·0						
Various	7·0						
Total	50%	28%	16%				

This is a point which is very difficult to determine. The vast majority of cases coming into hospital or treated in private practice who suffer from gastric ulcer recover, or are lost sight of, and it is exceedingly difficult even to make an approximate estimate of the ultimate result; but I lay before you some of the figures that have been brought out by those who have given most attention to the subject, and you will see that there is a fair amount of correspondence among them. Gerhardt, who is a well-known authority in these matters, gives the number of deaths from bleeding from gastric ulcer at from 3 to 5 per cent. (Table XX). Welch, who has also given a great deal of attention to the subject, states the deaths from bleeding from gastric ulcer at 3 to

great many surgeons have attempted to save patients who are suffering in this way from acute hæmorrhage, but very few have succeeded. The reasons are fairly easy to understand. The patient has been exhausted by hæmorrhage, and the operation has been exceedingly difficult and protracted, and even if the bleeding has been arrested the patient has been in a state of such intense shock that he has died from it. But some successful cases of this kind are on record, and they must be considered very brilliant achievements. One of the successful operators is Mikulicz, who, as you have seen, has had a very large experience in dealing with the stomach surgically. In one of his cases the coronary artery had been opened by a small round ulcer

on the lesser curvature. Laparotomy was done, the ulcer excised, and the opening thus made into the stomach afterwards stitched. The patient was alive and perfectly well several years afterwards. Roux, again, a Swiss surgeon, saved one sufferer who had had two attacks of severe bleeding. The patient was in a state of extreme anæmia due to the loss of blood. The ulcer was found to have opened the coronary artery on the lesser curvature. The necessary operation was as difficult to do as any in surgery. Then Küster had two successful cases of the kind. His two patients were practically bleeding to death. He opened the stomach and burned the base of the ulcer with the actual cautery, and then in order to give rest to the eschar which was thus formed, and, generally speaking, to the stomach, he did a gastroenterostomy straight off in both cases. Both patients were saved. These are the only cases of this particular kind that I have been able to find in which recovery followed operation for dangerous acute bleeding. In such instances of acute hæmorrhage the great difficulty is to decide which cases are severe enough to justify such an exhausting operation; and besides, we know that in many cases when the bleeding has led to syncope it stops of itself, and a clot forms in the injured vessel. In these cases the hæmorrhage may never begin again. But we cannot be quite sure that such a result is going to follow. As some of you know, two of my cases have lately died of hæmorrhage which we could not control medically, the patients having been killed by a perforation straight into an artery. Again, it is difficult to decide whether the individual is strong enough to bear any surgical treatment at all, and if it is undertaken too late it is worse than useless. However, the more general knowledge that surgery *can* cure such cases will lead to our being ultimately able to form a better judgment on the question of surgical interference; at present we have little definitely laid down to guide us, and therefore each case must be treated upon its own merits. Here of course medicine and surgery must go hand in hand. Physicians see a great many more of these cases than we do, and their experience will enable them to estimate the chances and probabilities in such cases, that is, how far the bleeding may go on without death occurring, and what are the chances of the hæmorrhage stopping. Moreover

physicians are constantly employing various drugs and applications in the endeavour to arrest this bleeding, and they know how much to expect from these remedies. In this whole subject of gastric surgery it is quite clear we never can divorce medicine and surgery.

It should also be emphasised that in many cases where surgeons have opened the stomach and searched for bleeding points they have failed to discover them. The patient has been bleeding violently, but yet the spot furnishing the blood could not be found. That is not difficult to understand when we remember that the point may be very small to the naked eye, and yet it may furnish a large amount of blood. Moreover the wounded vessel may be seated near the cardiac end, or in the lesser curvature; and there is no blame to a man if, when he has only made a moderate opening in the wall of the stomach, he fails to find the point from which the blood is coming.

We will now leave the group of emergency operations and pass to the consideration of GROUP II, that is, operations which are *remedial* or *curative*. Under this heading will come resection for chronic gastric ulcer; resection for pyloric ulcer, divulsion for pyloric stenosis, gastroplication for gastric dilatation, gastrolisis for the removal of adhesions, pyloroplasty for pyloric stenosis, and gastropasty for the hour-glass stomach, and then gastroenterostomy for all the above conditions. What these latter are you will see summarised in Table XX.

Table XXI (various sources).

Percentage of Results of Medical Treatment of Gastric Ulcer.

	Debove and Rémond.	Welch.	von Leube.
Recovery ...	50%	85.0	74.1
Improved	21.9
Unrelieved	1.6
Deaths	50%	15.0	24

University College Hospital. Cases treated in the wards from 1872 to 1890 = 200; deaths 15.

I have compiled it from various sources. It is of course necessarily very incomplete, for different observers have directed their attention to different

points of the subject, but by combining the figures you see that the serious consequences of gastric ulcer are many, and go far beyond mere fatal perforations or hæmorrhage. It is a formidable list—hæmorrhage, pyloric stenosis, inanition, pylephlebitis, cancer starting in ulcers, tuberculosis favoured or led up to by the inanition and the anæmia, and various other ill effects. Now if you look at the views of the various observers from whose writings I have compiled that table you will see they differ very widely. Debove and Rémond after analysing 100 cases came to the conclusion that 13 per cent. died of perforation, 5 per cent. hæmorrhage, but these latter writers do not mention pyloric stenosis at all. The latter condition, however, is credited in Gerhardt's list with 10 per cent. mortality. And in one of the most recent books on this class of diseases, that by Boas, he states that probably pyloric stenosis is the most frequent danger of all from gastric ulcer. Then comes inanition. We must admit that many of these patients must die of gradual starvation, using the term in its widest sense. Debove and Rémond put the deaths from this cause at 5 per cent.; pylephlebitis they do not mention. But Welch considers inflammation of the veins of the liver causes death in 5 per cent. of the cases. Cancer is not mentioned by any but one of the authors cited in my table. But Hauser came to the conclusion that when cancer is present in the pyloric end of the stomach, in 5 per cent. or 6 per cent. of the cases it has started in a chronic ulcer and caused death. Debove and Rémond put down the deaths from tuberculosis very high indeed, namely, 20 per cent. But that, I think, will be questioned by most observers.

I give you these figures for as much as they are worth, but all these writers are considered careful observers. You will then see from the table before you that the total percentage of deaths varies from 16 to 50 per cent. But the other night I took the records of our own hospital, and went through nineteen years. I found exactly 200 cases which had been treated in the wards for gastric ulcer, and of these only fifteen died in hospital. The cause of death of these fifteen I have not yet had time to make out; but the figures would give us only $7\frac{1}{2}$ per cent. of deaths from all causes while in hospital. What we require to know is the exact risks and disabilities, imme-

diate and remote, of 1000 patients suffering from well-marked gastric ulcer under observation for many years. As bearing upon that point I may show you what percentage of complete recoveries under medical treatment are supposed to take place by various writers (Table XXI). Debove and Rémond put them down at 50 per cent., Welch 85 per cent. (which is perhaps too hopeful), and Von Leube, who is believed abroad to be one of the greatest authorities on diseases of the stomach, estimates the number of recoveries at 74.1, those that are improved 21.9 per cent, and those that are unrelieved 1.6 per cent.; that would be a total of 96 per cent. of ultimate recoveries, and he estimates the deaths at only 2.4 per cent. of all cases treated in the wards. This authority claims to have treated 443 cases in hospital in a given time, and the figures I am dealing with are from the collected records of these cases. There is always a little difficulty, however, in accepting statistics like these where the physician or surgeon has become a specialist on any of these obscure diseases, because he is a little prone—I do not know that he can help it—to regard many obscure affections as coming into his own branch. I should suspect that many of these cases put down definitely as gastric ulcer, and treated as such, would not be recognised in this country as such, but would be considered as forms of aggravated dyspepsia without ulcer. In a very large proportion of these cases, however, he says there was definite hæmorrhage from the stomach, and his own belief, at all events, is that they were all definite cases of gastric ulcer.

But on combining these various tables it is evident at all events that very serious and wide-reaching consequences do follow gastric ulceration. This must be also clear from a study of any of the recognised writings upon the subject, and that is what we have to bear in mind. This is even more important in considering the last branch of our subject, namely, what I venture to call here *preventive* operations (Group III).

Now it is admitted that under suitable treatment the vast majority of patients suffering from gastric ulcer get well in the hands of the physician. But it is equally admitted that though there are a certain number of people who are in no great peril from gastric ulcer, there are also many others who, if not in actual danger, are doomed to live a

life of suffering from the condition or the disabilities consequent on it, which we may group under the heading of severe chronic dyspepsia. Those suffering from pyloric stenosis illustrate this very well. So do others who lead a life of pain due to adhesions, and there are many of these, as Dr. Roberts tells you. Moreover, weakness and chronic anæmia in these cases render the patient more liable to intercurrent affections, such as tuberculosis. It is for the relief of this class of invalids who have not been improved by medical treatment, be they few or many, that the surgeon is now being appealed to. Now Mikulicz, in the tables which I have given you, comes to the conclusion that there are risks to patients' lives in about 25 per cent. to 30 per cent. of those suffering from these forms of chronic gastric disease

different; there is a difference in regard to the danger, as you have already seen from the earlier statistics on the subject, and there is a wide divergence as regards the operation. In the more recent statistics this comes out better than in the tables of an earlier experience which I have given you, and the figures are larger, and therefore much more reliable. In studying these newer tables three striking facts come out prominently: first, that there has been a greatly lessened mortality from all operations on the stomach within the last few years; secondly, that the improvement is most marked amongst those performed for chronic non-malignant disease; and lastly, that an increasing number of such cases are operated on every year. The series of cases I now show you (Table XXII, Mikulicz) include the results at three different

Table XXII (Mikulicz).

*Results up to March, 1897, for Operations for Non-malignant Disease without Serious Complications.
Three Clinics.*

Operator.	To end of 1890.		1891 to October, 1896.		October, 1896, to March, 1897.		Together.	
	No. of Cases.	Mortality.	No. of Cases.	Mortality.	No. of Cases.	Mortality.	No. of Cases.	Mortality.
Billroth	15	60.0	5	20.0	20	50.0
Czerny	7	42.8	12	16.7	19	26.3
Mikulicz	5	20.0	21	19.0	10	...	36	13.9
	27	48.1	38	18.3	10	...	75	26.7

While going to press Mikulicz notes that since writing above his figures extended to eighteen consecutive cases without a death.

with or without ulceration; but let us put them down at 20 per cent., which I think is a fair estimate, and these are the cases we have to bear in mind when the question is put, "Can anything be done by operation for chronic gastric ulcer and its complications?"

One of the first questions put to us in relation to this whole subject of surgical interference is, "What is the risk of operating on the stomach in such cases?"

We have tried to form an estimate of the risks of operation for malignant disease, and also for the more acute non-malignant conditions; let us now try to form some estimate of the risks of operating on the stomach for the more chronic sequelæ, for the two classes of cases are quite

clinics all practically working upon the same lines. These figures are therefore far more reliable than anything you can collect from ordinary publications.

Note, too, that in the last six months of the year in which the paper from which these tables are taken was written, and while he was correcting for the press, he was able to state that he had operated on eight more cases, not one of whom had died, making a series of eighteen consecutive operations without a death.

Table XXIII, again, is a collection which he made, including all the records available, contrasted alongside with those which you have just seen. He was able to collect 238 altogether. You see here that the numbers who have sub-

mitted to operation for these conditions are increasing, and that the troubles for which operation was sought included open gastric ulcers not complicated, pyloric stenosis, hour-glass stomach, adhesions to adjacent parts, and pyloric strictures due to burns caused by caustic fluids having been swallowed. But the large number collected from the records of the three clinics arranged under the

years the number of resections was very small, while the other operations had greatly increased in frequency. And though the mortality in all the operations had fallen enormously, that of the two minor procedures showed the greatest improvement by a long way.

But if we take the most recent records of all from the experience of two operators uncompli-

Table XXIII (cases collected from all sources).

Results of Operations for Non-malignant Disease without Serious Complications.

Operator.	To 1886.		1887 to end of 1890.		1890 to end of 1896.		1896 to beginning of 1897.		Together.	
	No.	Mortality.	No.	Mortality.	No.	Mortality.	No.	Mortality.	No.	Mortality.
Billroth } Czerny } Mikulicz }	8	62.5	19	42.0	18	22.2	31	10.0	75	26.7
Other	15	33.3	31	34.8	87	18.4	30	10.0	163	19.4
	23	43.3	50	32.0	105	19.0	61	10.0	238	20.8

This table includes cases operated on for—

1. Open gastric ulcers not complicated.
2. Pyloric stenosis and hour-glass stomach.
3. Cicatricial adhesions with adjacent parts often requiring resection of ulcers.
4. Pyloric stricture from burns by caustic fluids.

Table XXIV (Billroth, Czerny, Mikulicz).

Operations for Non-malignant Pyloric Stenosis and Gastric Ulcer without Complications.

Operation.	To end of 1890.		1890 to beginning of 1897.		Together.	
	No.	Mortality.	No.	Mortality.	No.	Mortality.
Resection.....	28	39.3	18	27.8	46	34.8
Gastro-enterostomy.....	23	43.5	68	16.2	91	23.6
Pyloroplasty } and Gastroplasty }	21	23.8	52 } 14 } 76	33.2	97	15.6
	72	36.1	136	16.1	234	22.2

- Showing—1. Diminution in number of resections.
2. Increase of smaller operations.
3. Diminished mortality.

various operations (Table XXIV) show this more forcibly, and also the gradual change in feeling which has occurred in regard to the desirability of the various procedures.

In regard to this last point it may be seen from Table XXIV that up to the end of 1890 resections preponderated considerably over pyloroplasty and gastro-enterostomy, while in the following seven

cated by anybody else's work besides their own—as, for instance, those of Mikulicz (Table XXII) and Carle (Table XXV)—we find 18 operations for non-malignant disease by the former without a death, and Carle, of Turin, had only a percentage out of 41 cases of 11 per cent. mortality.

For pyloroplasty alone the mortality was, as you see, 15 per cent. in Mikulicz's collection of cases,

and for gastro-enterostomy with 91 cases you have 23·6 per cent. Carle, with 14 cases of pyloroplasty operated on by himself, had a mortality of 7·0 per cent., and in 27 cases of gastro-enterostomy only 3·8 per cent. Moreover you note that during the last two years, operating with Murphy's button, Carle has had 23 gastro-enterostomies without a single death.

Table XXV.

Carle and Fantino's Comparative Statistics.

Sources.	Pyloroplasty.		Gastro-enterostomy.	
	No.	Mortality.	No.	Mortality.
Haberkant ...	51	21·0	44	25·0
Woelfler	50	26·0	25	20·0
Mikulicz	97	15·0	91	23·6
Doyen	3	66·6	40	20·0
Carle	14	7·0	27	3·8

In the last two years, during which Carle has used Murphy's button, he has done twenty-three gastro-enterostomies in non-malignant cases without a single death.

From all this we get some idea of the mortality of the most recent operations, and we find it is brought down to about 10 per cent., and this knowledge enables us in some measure to estimate in cases of chronic ulcer without acute complications whether surgery should step in or not.

Let me try now to give you an idea of the various *remedial* operations for non-malignant disease, and endeavour to point out which ought to be selected.

Taking resection first, partial or complete, it may be said that it is practically abandoned in favour of milder operations. Loreta's digital divulsion is also almost given up; it is found to have a mortality of 38 per cent., and it is superseded by less dangerous operations giving more relief, and not likely to be followed by recurrence of stenosis. Gastroplication may also be put aside; it is unscientific and directed against a symptom only. The dilated stomach may be reduced to any extent by forcing, but that does not cure the disease.

Then we come to gastrolisis, or the freeing of adhesions. These latter may take place to adjacent parts, liver, spleen, gall-bladder, abdominal wall, and of course are due to chronic inflammation. The adhesions, because of the movements of the

stomach, often produce intense pain, and in some cases drag upon the organs to which they are adherent, and produce derangement of their functions. Now there are two classes of these lesions that you must keep apart in your mind's eye. Those cases of trifling adhesions which produce only pain and are very inconsiderable, interfere but little with the movements of the stomach, and are not dangerous from other causes. Then there are the very severe cases where the adhesion to the parts around has given rise to suspicion of malignancy, and so on, and which call for excision. As far as I can see we have only had a few cases operated on for adhesions in this country. Mr. Mayo Robson, indeed, has recorded some operations for adhesion between the gall-bladder and the stomach, and there are a few others, but not many. Abroad we find several such cases of great interest, such as those of Rosenhain, Hahn, Bardeleben, Terrier, Lauenstein, Riedel, Landerer. But let us take for instance the first successful one recorded by Billroth. In this he thought at first he was dealing with a malignant tumour adherent generally, but ultimately he found a mass of hard fibrous tissue round an ulcer, and excised a piece of the anterior wall of the stomach, stitched up the resulting opening, and the patient six years afterwards was in excellent health.

Another case operated on by Mikulicz is also on record, where a large piece of the stomach, including an ulcer and adhesions, was excised, the patient recovering perfectly, marrying and bearing children.

But the most remarkable case of the kind illustrating what the stomach will stand (perhaps these very serious cases are less likely to occur in this country, where the feeding of the poorer classes is better) I will briefly outline and read to you. This was also suspected, before the operation was begun, to be one of malignant disease. There were certainly adhesions to the abdominal wall, and on cutting down to the abdomen these were found with wide induration of the gastric substance. The operator, Hofmeister, then discovered the condition to be due to an ulcer, and he resected the piece of the stomach including it and the surrounding induration, which he describes as half the size of a cheese plate, involving the greater curvature of the organ.

In taking away this portion of the stomach and adhesions the operator had to remove a piece of

the liver. On the left side he also found massive adhesions running to the spleen; he then cut away a portion of the latter, and with it a piece of the abdominal wall. The wound was stitched up obliquely and the patient recovered perfectly, and was well when he wrote three months afterwards, and had put on thirteen pounds in weight and was eating what he liked.

Another case, and yet another which I need not go into, make some five of these very serious cases, forming a group in which the operations have most probably saved the patients' lives, and have certainly delivered them from a most terrible amount of suffering.

Now we turn to pyloroplasty. This operation is done for contraction of the pylorus through ulceration or chronic inflammation, and it has been performed many times; the mortality, as I have already shown you in these tables, ranges from 15 to 7 per cent. It appears, however, that this operation is also likely to go into the background; there is a tendency to replace it by gastro-enterostomy, which is a much easier procedure, and, judged by our statistics, is one which appears to have a lower mortality—a consideration that would above all influence us very much. The reason why gastro-enterostomy for pyloric stenosis due to ulceration is considered best is that besides being easier it is considered also to be quite as efficacious, since the stomach is thereby regularly emptied of its contents, which before could not pass through the stenosed pylorus. As the stomach empties itself the ulcer heals, and in many cases a considerable amount of spasm of the pylorus due to the irritation of the muscle subsides, just as in the case of a fissure in the anus; and a very small ulcer will produce this condition. The operation relieves the stomach, the patient gets stronger from absorption of food, and the ulcer heals up, and the pylorus may again become freely pervious.

You will therefore probably hear in a few years that pyloroplasty has receded into the background. To be sure, if it is done successfully in cases where there is not much ulceration it reproduces the *status quo ante*. You have no risks from actual closure of the part, but it appears to have a higher mortality, and it also has been followed occasionally by recontraction. Gastro-enterostomy, on the other hand, the mortality of which has been reduced by one operator to 3·8 per cent., is now

becoming the procedure *par excellence* for remedying these defects; the operation is done by a great many surgeons on the anterior wall of the stomach by Woelfler's method, or on the posterior wall by von Hacker's, and undoubtedly it has given in many cases great relief, and some of these operations have been followed for many years by good health. But Carle, who has up to the present had the best published results in these cases, is a strong advocate of the posterior method, and advances very cogent reasons for his preference. And I will remind you again of the fact that of his last 23 operations, done with Murphy's button through the meso-colon and the posterior surface of the stomach, he has not lost a single case.

IN tuberculous joint affections the conservative tendency of German surgeons is very striking. Operative measures are put off until it is perfectly evident that no less radical means are of any avail. Injections of iodoform emulsion ten per cent. are made, not once or twice, but three, four, and sometimes five times, in the hope of halting the progress of the disease without recourse to the knife. A popular bit of surgical technique in this matter just now is the taking of a Roentgen photograph of the joint just after the injection of the iodoform. As the salts of iodine, like the salts of the metals, are opaque to the X rays, a very good idea of the distribution of the iodoform is obtained by this means. As the drug is not soluble in the fluids of the joint, and inhibits bacterial growth only by its actual presence on the surface of diseased tissues, it is important that it should be thoroughly distributed over the internal capsular surface and the bony cartilages. As ordinarily injected, however, it needs but a small fringe of diseased tissue to so alter the direction of the injected liquid that most of the emulsified iodoform will be deposited over but a very limited area. The subsequent passive movements and kneading of the joint will only partially overcome the defect of technique. Time is precious in the therapeutics of such cases; the bacillus is ever being buried more and more beneath inflammatory products, out of the way of antiseptics, so that the Roentgen procedure would seem to be a promising one.—*Therapeutic Gazette*, April, 1898.

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A DEMONSTRATION OF CASES AT THE CENTRAL LONDON SICK ASYLUM,

March 24th, 1898,

By GEORGE THIN, M.D.

LADIES AND GENTLEMEN,—The first case to which I direct your attention is one of rodent ulcer in a woman æt. 71. Nine years ago this woman had a small swelling on the left cheek, which ulcerated after two years, but was not shown to a medical man until three years after its commencement. It was then the size of a farthing, and did not penetrate the cheek. It has steadily spread, and during the last twelve months there has been severe pain at night. On admission in July, 1896, the area of ulceration embraced two-thirds to three-fourths of its present extent, and going backwards from its posterior border there was a hard ridge in the skin, which terminated in a thickening behind the angle of the jaw. This ridge is now broken down by ulceration. There have been several attacks of local inflammation in various parts of the body, with much swelling and redness, and pain and tenderness apparently in the cellular tissue, which have terminated without suppuration. During some of these attacks the temperature rose to 104°. You will see how very extensive the ulceration is now, having eaten away practically the whole of the left cheek. But extensive as it is, you will notice if you regard it carefully how entirely local the affection is. It does not spread deeply, and there is no infiltration; it is almost as clean as if the tissue had been cut out with a scalpel. The differential diagnosis in this case would not be difficult to an experienced person. The first point is, how would you distinguish it from ordinary epithelioma? In a case of epidermic epithelioma you would certainly have a hard infiltrated edge outside the red ulcerated line; that is a great point of distinction between the two conditions. Although in rodent ulcer there is a great destruction of tissue, there is

nothing producing any very great inflammation, whereas in epidermal epithelioma there is always a considerable amount of infiltration in the cellular tissue round about, with redness and swelling.

Now, it is a very important thing to understand the beginning of this disease. A small swelling appeared on the cheek, which did not ulcerate for two years, and she did not show it to a medical man for three years, proving how very little inconvenience it must have given her. If it had given her pain she would have sought advice for it, and if it had looked bad she would have done so. If this patient had consulted a medical man for the condition any time during the first two years, and if it had been excised sufficiently widely, you would not have now seen this ulceration. I have seen frequent cases of this kind, recognised at a sufficiently early stage, excised and the edges of the wound brought together, resulting in perfect success and permanent cure. I can look back and remember many patients who have had no recurrence. Rodent ulcer, which has been a bugbear in surgery for so many years, as a rule is not recognised sufficiently early.

Some twenty years ago I examined histologically a rodent ulcer which was excised by Sir James Paget from the back of a man. At that time it was a large rounded ulcer about two and a half inches by three inches, and had taken forty years to reach that size. For the first twenty to twenty-five years the growth made scarcely any progress. It was freely excised by Sir James Paget. The man died some time afterwards of an intercurrent malady, but there was no recurrence of the tumour.

I remember another case of a lady who consulted me eight or nine years ago with an ulceration, which was considered to be epitheliomatous, in the centre of the right cheek. The ulceration was very much the same as this woman describes hers to have been in the early stage. I asked my friend Mr. Watson Cheyne to freely excise the ulcer, which he did, and there has been no recurrence. I can remember other cases in which early diagnosis has resulted satisfactorily. The important points about the disease are, first, to diagnose it early, and, secondly, to freely excise it. If there is a small raised pimple, particularly if it shows a tendency to have a border, occurring in an elderly person of fifty or upwards, slowly increasing in size, it should be looked upon with great suspicion.

If it be partly transparent, having a semi-fluid consistence, that adds to the suspicion that it is the early stage of rodent ulcer. But unfortunately medical men seldom see these cases before they ulcerate. In even advanced cases of this disease, if the condition permits it, free excision around the border and the subsequent bringing of the parts together again always gives a chance of recovery. If the ulcer is in the upper part of the face there comes a time when it may get into the cancellated tissue between the plates of the skull. Once it gets there it spreads with great rapidity. Then it runs the same course as other morbid growths do in the same parts.

Case 2.—E. A.—, æt. 85. This patient is an old woman with a scirrhus tumour in the breast, which is hard, but not very large. What I particularly want to direct your attention to is the appearance of the skin over the tumour. There is a well-defined area of red skin, cleanly bordered, but without a raised edge. There is apparently no discharge from the rather rough leathery surface. This condition is due to the pressure of the tumour, which obliterates the blood-vessels, and causes the tissue to undergo a slow necrosis, a different condition of things to that observed in the large fungating, infiltrating, and inflammatory tumours often associated with cancer. The condition of the skin is such as to lead you to the diagnosis even if you felt no tumour. This lump appears to have been there a year or more. It does not grow much, but it adheres to the skin, and moves a little over the subjacent parts. But it does not retract the nipple. Very large glands can be felt in the axilla. You can see that there are certain thoracic symptoms, dulness at the right base, and an abrupt cessation of breathing. There are moist râles. The local symptoms are not of much importance comparatively considered in connection with the general condition—a fact due to the patient's age.

I have now to invite your attention to Case 3, that of a woman æt. 49, a very interesting case of erythema dependent on disease of the nervous system. I ask you to notice the pink and mottled condition of the skin. When she was first admitted, eight or nine years ago, there was every evidence of her having had an attack of cerebral hæmorrhage, and for many months afterwards she had paralysis of the sphincters, and mental weak-

ness. She has remained in a peculiar torpid state ever since. Her limbs are stiff and wooden, and there is little variation in her expression. Her mind is easily confused, and her understanding very limited. There is no paralysis, no excessive reflexes, and no loss of sensation. But she has this mottled, purplish erythema, which varies in intensity from time to time. You will see that her feet are the colour of dark plums. The nails are very small and have but little depth, and the fingers and toes appear stunted. The point about this case is that these changes which you see caused by a disordered action of the blood-vessels are clearly due to the state of the nervous system. Her nervous system has been more or less under the influence of the sequelæ of the cerebral hæmorrhage ever since it occurred. The portion which is now at fault is clearly the centre which controls the vaso-motor nerves.

There is a disease of which you do not hear much in England, which was described many years ago by Bazin under the name of "*erythème induré des scrofuleux*," which is mostly found on the legs below the knees, and chiefly, but not exclusively, in young women. It is very common in women who stand much, such as in washing, and who, while in not very good health, become very fatigued. In these cases there are frequently found patches of this erythematous condition one to two inches in diameter, the colour resembling very much what you see on the back of this woman's foot. In this affection you often get a certain amount of ulceration, and an interesting point about this ulceration is that in many respects it looks like the ulceration of tertiary syphilis, and has often been mistaken for it. Dr. Colcott Fox in this country has studied the disease very carefully, and has published a paper on it which is well worth reading, and Mr. Hutchinson has also written on the subject. We know that loss of nerve power can cause both erythema and ulceration; in nerve leprosy, for example, in which the disease may go on to perforating ulcers. A case of the kind we are now considering, occurring not only in the lower limbs, but also in the hands and other parts, in a patient who is markedly suffering from nerve lesions, throws considerable light on the other class of cases of an analogous nature. It is clear that mere standing could not produce permanent

patches of erythema on the legs and wrists of a healthy person, and you must have something else as a causative factor. Bazin's disease is very probably due, though that is not proved, to a suspension of the nutritive function, such suspension being due to defective innervation. This case has interested me greatly for that reason. Then in addition to the erythema there is the shrinking in the phalanges of the fingers and toes which I have alluded to; one of the bones of the little finger is clearly shrivelled to about half its normal size. In nerve leprosy you often get a shortening of the phalanx, which still remains covered with skin, and with no breach of surface. Indeed, you may, in that disease, get an actual amputation of the finger without breach of surface, the bone becoming absorbed altogether. I saw a man in the Whitechapel Infirmary some years ago suffering from well-marked nerve leprosy, in which the whole of one of the metatarsal bones of a foot had absolutely disappeared, simply shrinking from the loss of nerve-supply. That is the lesson to be derived from this most instructive case.

Case 4.—The next patient, a woman æt. 63, presents an interesting example of leukoderma. She has had four children, but can give no definite account of the condition of her skin beyond the fact that it has been as you see it for a long time. You will observe that there is increased pigmentation of the skin, with leukodermic patches, in which there is a tendency for the pigmentation to decrease. The pigmentation has appeared in nearly symmetrical areas, roughly following the distribution of the cutaneous nerves in the lower part of the abdomen and down the outsides of the thighs. Her health is not good: she is anæmic and suffers from hæmorrhoids, and has a mitral regurgitation. The colour of the part I show you somewhat resembles that of tinea versicolor, but it is not that condition. The affection is strictly limited to the abdomen and outer part of the thighs.

If you examine this coloured skin carefully you will see that it is not finely scaly, like that in tinea versicolor. Possibly in a country where leprosy was common such an appearance might be thought to be connected with nerve leprosy, but against that is the distribution and the absence of anæsthesia. Any one at all familiar with nerve leprosy should not mistake one for the other. What gives rise to this condition? Why should a naturally

healthy skin first of all become pigmented and then free from pigment? Our knowledge of the pathology of that affection is chiefly due to a distinguished French physician who recently died—Professor Leloir—who examined the tissues and nerves histologically, and was able to demonstrate a degeneration in the nerve-fibres. The brown patches themselves are found to contain the first stage of the disease. As the disease progresses the epidermis becomes thinner, in addition to the pigment being absorbed. A very curious thing shown in this disease is that if profuse perspiration is produced by the injection of pilocarpine the healthy skin acts in the usual way, but the white patches of skin do not sweat—or if they do sweat at all it is very little,—further showing that it is a nerve disease.

I was consulted by a patient hailing from the West Indies who was supposed to be suffering from leprosy, but it turned out to be simply a case of leukoderma of the hands, and the white patches had been mistaken by some one not cognizant with leukoderma for the graver disease. You find pigmented patches come in the skin from various affections of the nervous system. They have been known to follow a blow on the nape of the neck, when there may be pain and general nerve irritation, followed by patches. Pigmented skins are very common in insane people and among epileptics. Pigmentation may also follow local nerve lesions. They may thus arise in two ways, namely, from a central nerve lesion or from a local nerve lesion. After severe facial neuralgia there may be such patches about the face. The pigmented forehead sometimes seen in pregnant women is no doubt due to an affection of the cutaneous nerves associated with the pregnancy.

Case 5.—This man presents a very remarkable condition, which well deserves looking into. He acquired syphilis thirty years ago, and he has suffered very much from eczema since November, 1897. There is a large area on both legs presenting a serpiginous border with large papules, and a zone of deep pigmentation with a centre of leukodermic skin. His tongue is devoid of papillæ at the tip. He has got eczema of the forehead, and has albuminuria. He has lately suffered very distressing hardships, having had to walk in the extreme north of Canada a distance of 1600 miles. You will see that the cutaneous irritation

this man suffers from is simply a hyperæsthesia of the skin, which is called into play when it is exposed to the air, but this condition itself would not give rise to much irritation. Syphilitic affections do not itch; but here is a specific affection occasionally accompanied by extreme itching. The itching cannot be attributed to the specific disease; you must bear in mind the man's albuminuria and his eczematous tendencies, and the extreme privations he has undergone, which probably would have brought on this pruritus of the skin apart from syphilis altogether. If you want to find whether an eruption itches you must look carefully for scratch marks. Here we do not see many. You must consider the case as one of specific disease, the manifestation of which is largely modified by the man's general state of health.

Regarding the treatment of these tertiary conditions, I should like to tell you something from my own experience. We know that the one remedy is iodide of potassium, but you find cases in which iodide of potassium alone does not act; but if combined with tonic treatment, particularly treatment by iron and other tonic minerals, we find healing often takes place within a short time. I can recall several cases of this kind. One man had a tertiary eruption on his back, partly ulcerated, and apparently simply swollen and indurated, which refused to yield to twenty grains of iodide of potassium three times a day, but when this was continued in combination with tonic treatment the condition began to heal immediately. What can you do in a case of syphilis in which iodide of potassium cannot be borne? Such cases are not common, but they occur.

I was once asked to advise in a case of specific disease of the cheeks and part of the nose, producing much deformity, which a distinguished physician had been treating three or four years. This patient had a special idiosyncrasy against iodide of potassium. He had been told that he must take iodide of potassium in a very small dose if he could not stand it in a large one. The dose which it was found he could tolerate was half a grain three times a day, but of course this did not cure the disease. I advised him to take twenty grains of iodide of potassium three times a day. He said he could not take it, but we sent him down to Devonshire to live in the country and to swallow

the dose of iodide three times a day, happen what may. At first it made him very ill, but afterwards he began to tolerate it, and in a remarkably short time the syphilitic ulceration healed over and became as well as such a thing could become, considering the loss of substance which had already taken place. There has never been any relapse. Benefiting from that experience, I have often given iodide of potassium notwithstanding protests of intolerance, and have advised patients in these urgent cases to take it whatever they may suffer from it. It is a curious fact that large doses of iodide of potassium may sometimes be borne when small doses produce irritation.

Case 6.—The next patient is a woman with specific eruption on the face and scalp. On admission there were several circular patches on the face and scalp, with papular borders and invading the nostrils and corner of the mouth. There is considerable pigmentation, and the skin of the legs is thickened. She at first had iodide of potassium, increased to half a drachm, three times a day, with perchloride of mercury outwardly in the form of ointment, but improvement only took place when this was given up and precipitate ointment applied. There is congestion of the nose, with a tumefying swelling, but no very well-marked border. You will see that there is also a syphilitic sore on the front of the knee. It was covered with a thick crust when I first saw it, and the effect of the treatment has been to take off that crust. This throws great light on the condition of the face, where the nature of the condition would otherwise be puzzling. The diagnosis could be arrived at by a process of exclusion. In diagnosis, when you see these breaches of surface you have to bear in mind three things—syphilis, cancer, and lupus, the three granuloma diseases; and it is very often possible to diagnose a case by excluding one or two of these. Perhaps the iodide of potassium began to benefit this patient after it was given up. Sometimes we do find benefit from the drug after its administration has been left off.

Antitoxine and Diphtheria.—By Dr. H. J. Patton ('Med. and Surg. Rep.,' January, 1898). The author, speaking of antitoxine in diphtheria and true croup, comes to the conclusion that the use of antitoxine is equally as efficient in true croup as in diphtheritic croup, yielding results even more readily in true croup.

WITH DR. MURRAY IN THE WARDS OF CHARING CROSS HOSPITAL,

February 24th, 1898.

LADIES AND GENTLEMEN,—This case is an instance of the mildest form of Raynaud's disease. The patient is aged 21. She has always lived in London, and has always been a total abstainer. She was perfectly well till August, 1894, when she first noticed that the index finger of the *left* hand "went numb," a tingling sensation being experienced at the time. On each occasion the condition only lasted for a few minutes. A little later—the exact date she does not remember—the middle finger, and then the third and fourth fingers of the same hand, suffered in the same way. The condition lasted a little longer than at first, and sometimes would persist for two hours if the patient happened to be out of doors. The *right* hand was next attacked, quite a year after the left. Then the condition spread down the left hand, and a little further into the palm. Next the left foot became affected, and then the right foot. There was a distinct interval between the involvement of each limb. We are told that the tongue, too, has been affected. No one here has seen anything wrong with the tongue, but the patient is quite clear about the fact, and says the sensation was in every way like that experienced in the fingers. The usual rule is illustrated by this case, namely, that the condition comes on in cold weather, or if the hands are exposed to cold water or anything else cold. If the weather is milder the condition is milder. After an attack of numbness there is a burning sensation in the parts. You will observe that the anæmic area has a definite edge to it, though this afternoon it is not so distinct as usual. If you look at her fingers you will see that on the ends of them there are little pointed cicatrices. The explanation of these is simple. The ends of the fingers being so continually blanched have less power of nutritive repair than normal fingers, and also being cold they are numb, and therefore less likely to avoid injury. Into abrasions thus accidentally produced, the cocci on the skin grow. Small abscesses result; these are readily brought

about because of the defective resisting power resulting from the defective circulation. The cicatrices mark the position of these small abscesses. With regard to her other symptoms, one may dismiss them by saying that she has had nothing wrong with the urine or with the heart. There has apparently never been any hæmoglobinuria. Blood taken from the finger end at the time of a paroxysm is perfectly natural. In some recorded cases it was found that the blood-corpuscles were broken up. This breaking up of corpuscles occurred more especially in cases associated with acute pain or with hæmoglobinuria. The electrical reactions are important, because multiple neuritis has sometimes been made out; in this case, however, the reactions are normal.

As regards treatment, warmth is the chief thing that does good. It is that which prevents the condition coming on. The patient is clothed in flannel; her feet and hands are kept wrapped in cotton wool, and thus many of the attacks are warded off; but directly the parts are exposed the attacks come on again. Some time ago Dr. Bruce had a case in which he used morphia very successfully; at the present time this patient is taking twelve minims of the liquor three times a day. She has not been taking it many days yet, so it is impossible to forecast the result. The ordinary vaso-dilator drugs—nitrite of amyl and nitrite of sodium—are sometimes employed, but the results of their employment are not very encouraging. In the majority of cases they have failed. Electricity is spoken of very highly by some people, and I expect we have all seen cases in which its use has been followed by marked benefit. The positive pole should take the form of a large pad placed on the back or abdomen. The hands or feet are then put into hot water, and the negative pole is also placed in the water. The continuous current is generally employed, although sometimes the induced current does an equal amount of good.

Mitral Stenosis.—This boy is a typical case of mitral stenosis. There is no history of rheumatic fever, but this is no proof he has never had it. Five years ago he began to complain of dyspnoea and of copious hæmoptysis; these were his chief symptoms, and they have recurred several times since. At present he has a very pronounced

presystolic thrill and a loud presystolic murmur. The murmur is very characteristic; it runs right up into, and ends sharply with, the first sound—the first sound itself having that character which is nearly always found in comparatively early stages of the disease. It is short and sharp, resembling the second sound, for which it is often mistaken by students. This case is an apt illustration of the importance of timing the systole by the hand on the chest wall while you are listening to the sounds with the stethoscope. Unless this is done you are liable to mistake the short first sound in such a case as this for the second, and to infer, therefore, that the murmur which precedes it is systolic and not presystolic. You will notice from the displacement of the apex-beat directly outwards, and from the increased area over which you can feel the cardiac impulse, that it is the right ventricle which is hypertrophied. No distension of the left ventricle during diastole can occur, and as the resistance to contraction is therefore not increased, no enlargement of this ventricle follows.

The hæmoptysis in these cases is interesting. There is a certain similarity between it and the hæmatemesis which occurs in cirrhosis of the liver. In cirrhosis of the liver and in mitral stenosis the blood-stream meets with an obstruction, and the engorged vessels from time to time rupture and discharge part of their contents. But the prognosis in the two cases is widely different. In cirrhosis of the liver, hæmatemesis means that you are within sight of the end (two years or less). In mitral stenosis, on the other hand, hæmoptysis may recur over a far longer period without any such significance. This boy, for instance, has had hæmoptysis at various times for five years, and he may go on having it for another five. The reason why hæmorrhage occurs at an earlier stage in mitral stenosis than in cirrhosis of the liver is that the enormous strain put upon the lungs during coughing in mitral stenosis has no counterpart in cirrhosis of the liver, and that the mechanism of the distension is also more powerful in mitral stenosis. In this disease the heart pumps the blood forcibly against the obstruction, whereas in the portal region the blood is flowing much more easily and under a lower pressure. Of course the blood-vessels of the stomach are not better supported than those of the lung.

Convulsions.—This little girl is convalescent

from an attack of hemiplegia. Last November she was going to school, and on the way had a fit. She was brought home, and when she recovered from the fit the right side of her body was found to be paralysed. She remained in a condition of semistupor for several days afterwards. When she was admitted here, some five days after the attack began, she said "Yes" and "No" to most questions asked her, and her mental condition was "dull." It was impossible to say whether she had any true aphasia. In about a fortnight she regained a good deal of her power of speech, and could use the face and tongue, but the paralysis of her arm and leg persisted for a longer period. Even now—three months after the onset—she has very little power in the right arm and hand, and her leg drags in the typical hemiplegic fashion, although she can walk without assistance.

Probably the arm and leg on the paralysed side will not grow so well as those on the other, and a certain degree of talipes equinus will be produced, though it may not be more than can be corrected by a thicker sole and heel to her boot. I am going to use this case as a text for a few remarks on convulsions in children. In my student days convulsions were divided into three groups—sympathetic, symptomatic, and essential; the sympathetic being supposed to be reflex, the symptomatic to be due to organic disease, and the essential to be dependent on some unknown central nervous condition, or, in other words, to be epileptic. For practical purposes the best way is first to inquire whether there are present any unquestionably *predisposing* influences. First among these is *age*. Every one knows that convulsions are commonest among children between the ages of nine and eighteen months, and that they are also very common up to the end of the third year. After that they get less common. When *rickets* exists, it often acts as a predisposing cause. *Heredity* does not practically count for much. You cannot base a diagnosis on the fact that the father had epilepsy or was alcoholic or neurotic; still, heredity does play a certain part. Another very important predisposing cause of convulsions is *organic brain disease* itself in an *early stage*. Long ago Gowers insisted on that, and it is an important thing to remember. Meningitis or tumour may be present, but may not have given rise to any symptoms sufficient to have brought

the case under the observation of a doctor; and yet in such circumstances a little trivial reflex irritation may apparently cause a convulsion. In the course of the next week or fortnight symptoms gradually develop, and there may be fever, paralysis in some form, headache, vomiting, or optic neuritis. An important practical point in this connection is that the end of the simplest case of convulsions has not been reached until recovery is absolutely complete in every way, because a fit may be simply the initial stage of some serious disease. Then besides these four predisposing causes there are the ordinary exciting causes, the alimentary—improper ingesta and ascarides,—the dental, and the aural causes. I think we are rightly a little suspicious about the evidence upon which teething as a cause of convulsions in children rests. There are children who have had a convulsion, and one finds an inflamed gum. This is lanced, and there are no more convulsions. When we have said that, we have produced all the evidence that we can in favour of teething as a cause of convulsions. Age and rickets are the great causes of convulsions. All we can say is that gastric causes, dental causes, and even aural causes, such as otitis media, are insufficient to produce convulsions unless we have age or rickets or commencing organic brain disease existing at the same time. But there are also other causes. First of all convulsions may be the first symptom of the onset of an acute specific fever. That is one reason why it does not do to diagnose a convulsion too absolutely the first day. Convulsions may also mark the advent of infantile paralysis, either spinal or cerebral. Again, they may be the first evidence of uræmia, particularly in an unrecognised case of scarlet fever. You may say this is rather far-fetched, but I have seen two cases in which uræmic convulsions were absolutely the first signs that made the parents call in a doctor. In both cases the children had had scarlet fever, and were desquamating at the time they had the uræmic convulsions. Children in whom scarlet fever goes on unrecognised even by the parents are most of all likely to get Bright's disease. It is always wise in a case of convulsions to examine the hands and feet, to see if they are peeling. In whooping-cough sudden collapse of the lung may cause convulsions, and they may also occur as a result of the cerebral anæmia

accompanying syncope due either to post-diphtheritic paralysis of the heart or to sudden hæmorrhage. I have seen two children convalescent from diphtheria, and apparently doing well, suddenly taken with convulsions and dying in an hour. In such a case the heart fails first, the pulse gets very small and rapid, and the convulsion comes on in an hour or two, and as a rule ends fatally. Again, burns in children are sometimes followed by convulsions. With organic brain disease we must deal more fully.

We have spoken of organic brain disease as a predisposing cause, but it may also act as an exciting cause and produce convulsions. In an adult the convulsions due to brain disease—say of the cortical motor area—will very likely have a local *onset*, and may even have throughout a local distribution. The onset will always be the same at each attack. *Consciousness* will be more or less retained, and the part which is most convulsed is often temporarily *paralysed* afterwards. If these three things are present you are sure of your diagnosis. In a child you will rarely get all three. The convulsion will probably be a general one, but the onset may be the same in each case,—that is to say, may always begin in a particular part. Thus localised disease in the right motor area which in an adult would give rise to a convulsion limited to the hand and arm, in a child would more probably produce a convulsion beginning each time in the hand and arm, but spreading to and quickly involving the whole body. Of course consciousness in a young child is not easy to make out, and the temporary paralysis is not so often found in a child as in an adult. In the case of convulsions in a child you cannot at first be sure what the child has eaten; you cannot be sure of organic brain disease; and you cannot be sure of the onset of an acute fever. Other things you may dispose of, but these important ones you have to wait for, and nothing but waiting, and taking the temperature, and using the ophthalmoscope can make you absolutely certain of your diagnosis. A useful routine to adopt when called to a case of convulsions is—Watch the fit, its exact distribution, its intensity (whether very violent or whether mere muscular twitching), observe the colour of the patient (whether pale or cyanosed), and the degree of consciousness, and if practicable take the temperature in the rectum.

When that is done, ask questions, the age of the

child, the child's previous health—which, of course, includes inquiry into other fits and their nature, inquiries regarding acute specific diseases, sore throat, rash, whooping-cough, possible ingesta and conditions of the bowels, headache, giddiness and vomiting, and finally the exact character of the onset. In the meantime, according to the orthodox routine a warm bath (95°) would have been prepared. While the child is being undressed you look at its hands and feet and see if they are desquamating; then examine its chest, and see if it has rickets or anything wrong with the heart or lungs. Of course, if you find anything wrong with either of these you will not allow it to be bathed. After the convulsion has subsided look for any possible paralysis. After that, examine its ears, and see if there is a foreign body in either, or any disease. Examine the urine, too, for albumin.

As to treatment, if you do not come to any more definite diagnosis than rickets and slight peripheral irritation, bromides are useful, but they must be given in large doses. Five to ten grain doses may be given to children a few months old, and repeated. There is a practice nowadays of giving nitrite of amyl and nitrite of sodium, but I do not think they do any good. Chloroform inhalations are very strongly recommended by some authorities, and I have never seen them do harm. They reduce the intensity of the convulsion, and possibly tend to arrest it. Chloral may be used during the convulsion as an enema, and sometimes may be given by the mouth. But chloral is dangerous for a young child. Chloroform inhalations and chloral are both out of the question if there is anything wrong with the heart or lungs. If the fit is due to cardiac failure, stimulants must be given without delay. Next day see if the recovery is complete, and take the temperature, not forgetting, if practicable, an ophthalmoscopic examination.

Cerebral Gumma and Thrombosis.—This man is 58 years of age. Three months ago he had two fits, both on the same day. It is not very certain how long each lasted; but after they had passed off he was very violent, so much so that it was thought possible that he had commencing delirium tremens, or that he was the worse for liquor. Up to that date he had been perfectly well, but ever since he has complained of loss of memory and of intense headache, while

his wife adds that he has been "knocking over things" through clumsiness. Some six weeks after the fit he returned to work, but had to give up again after a day or two. Then he was sent to Hastings for a time, which I mention as showing that he was not at first bedridden. He took to his bed only ten days ago. There was no ascertainable history of syphilis, nor did examination reveal any characteristic appearances. On the 12th of this month (February, 1898) he became much worse mentally, and on the 15th he could do little more than answer simple questions. On the 17th he was extremely drowsy, much as he is at present. If told to put out his tongue, open his eyes, or hold up his right arm he did so; but we could not get him to speak. The left side of his face, the left side of his tongue, and the left arm were paralysed. The facial paralysis was of the usual hemiplegic type,—that is to say, the occipito-frontalis, orbicularis palpebrarum, the corrugator supercilii, and the dilator naris had escaped. His left leg was weak, but not absolutely paralysed. His knee-jerks were very marked, and he had slight ankle-clonus on both sides. His heart was weak, his optic discs normal, and his urine healthy. He had no vomiting, and was passing his motions and urine unconsciously. On the first and second days after he was admitted here he was less drowsy, could move the angle of his mouth a little, could protrude his tongue in a straight line and could talk, though most of what he said was incoherent. He has always recognised his friends, and he seemed at first to be generally improving, though he still passed his motions unconsciously. Two days ago, however, the left arm began to get rigid, and you will notice it is still so. He has slight degeneration in the arteries of his limbs. Of course there may be marked arterial degeneration in the brain, without the arteries in other parts being much involved. The best illustration of this fact I have ever seen in this hospital was in the case of a famous chess player. He had been over to America playing for a championship, and had done badly, to everybody's astonishment. He returned to England, and had not been back many days before he had an apoplectic fit just outside the hospital, and died of cerebral hæmorrhage. At the autopsy the arteries of his limbs showed little evidence of degeneration, while the degeneration of the arteries

of the brain was extreme. In making a diagnosis in the case before us we have to think of cerebral tumour, arterio-sclerosis, and thrombosis. It is certain that he has now thrombotic softening, and that it began about the 12th, but it is doubtful whether the earlier symptoms were due to a tumour or gumma, or to vascular disease. The absence of vomiting and optic neuritis are somewhat against the first suggestion, though the exaggerated reflexes are in its favour.

Arterio-sclerosis is a common condition, and it is likely that the loss of memory and the general failure to do his work depended upon defective blood-supply. Arterio-sclerosis leads to narrowing of the lumen of the vessels, and to consequent diminution of the blood-supply. When in this way the nutrition of the brain is lowered the highest functions suffer first. His memory therefore failed him before he began to have paralysis. At first when he began to improve after admission I thought there could not be any thrombosis, and that the nutrition to his brain was interfered with by the narrowing of so many branches at the same time. But this rigidity which has come on makes me revert to the idea that even at that time he had blocking of a certain number of vessels. I expect now that the condition will spread, and that he will rapidly get worse and worse.* Even if he does not, he will probably not regain any of his mental powers. When such cases do not go on to thrombosis, they often become inmates of our asylums; though mentally weak they may live for some years. The rigidity came on within less than a week of the thrombosis, if the thrombosis began at the time I have suggested. This is called "early" rigidity. There are two sorts of rigidity connected with the onset of cerebral disease. The earliest is that which comes on at the time of the attack, and is caused by the hæmorrhage as it ploughs its way through parts which are held tightly together. This form is common in hæmorrhage into the pons—paralysis and rigidity coming on simultaneously. Then comes this so-called "early" rigidity. It appears a few days after the onset, and is sometimes associated with a slight rise of temperature. That nearly always means either that the lesion has spread, or that some febrile or inflammatory disturbance is taking place

* At the autopsy a gumma with adjacent softening was found.

round it. In all cases it is a bad sign from the point of view of prognosis. In all probability this patient has only a few days to live.

Stricture of the Œsophagus.—This man is 55 years of age, and by occupation a brick-layer. He was quite well until June, 1897; he then began to complain of sickness. He had no pain, but he occasionally vomited directly after food. Then the vomiting became more frequent, and in October he came to the hospital. Towards the end of the year he began to vomit all the solid food he took, and even to complain of difficulty in swallowing liquids. During the whole of that time he was losing weight rapidly. He was still without pain, and if he limited himself to a liquid diet and took plenty of time over his meals he could swallow and retain what he swallowed. He noticed that cold fluids were much more difficult to swallow than warm fluids. Vomiting, when it occurred, took place directly after the act of swallowing, and the vomit was either neutral or slightly alkaline, and therefore contained no free hydrochloric acid. No blood or mucus had been seen in it. The symptoms obviously suggested the presence of some obstruction in the Œsophagus, and the question arose whether it was due to malignant stricture or to spasm. I am excluding fibrous stricture and cicatrisation because there was no history of his having swallowed any corrosive fluid, or of anything likely to have produced such a condition. Let us briefly look at the probabilities of the case as far as we have gone. First as to *sex*. Malignant disease of the Œsophagus is far commoner in men than in women, the proportion being about 75 per cent. and 25 per cent. respectively. On the other hand, spasm of the Œsophagus is much commoner in women than in men. Malignant disease is commoner at the *age* of our patient, while spasm is commoner in young people. Again, the *obstruction* in this case was *constant*. One of the most important points about spasm of the Œsophagus is its fitfulness. In many cases, if the subject of spasmodic stricture of the Œsophagus is upset, or frightened, or alarmed, nothing can be swallowed, and there is no help for it but to wait until the attack has passed off; moreover, swallowing is often noisy and accompanied by considerable eructation. Again, the *absence of pain* is in favour of malignant disease. Of course pain may be absent in malignant disease, but spasmodic

stricture without pain—pain generally of a neuralgic type—is undoubtedly rare. One point which may have suggested spasm is that the patient could not swallow *cold* liquids so well as those which were warm; this is a frequent symptom in spasm of the Œsophagus. The *emaciation* is an important consideration. It does not go for very much in the early stage, because it may depend solely on the inability to get food into the stomach. But if, after this difficulty is overcome, the man still loses weight, the diagnosis of malignant disease will be still more probable. In cases of spasmodic stricture patients will not lose flesh if they succeed in swallowing. When this is seriously interfered with they also lose weight. I remember the case of a clergyman who was reduced to a condition of considerable emaciation by spasmodic stricture. He was a very neurotic person, but he eventually got quite well.

Having by careful examination done our best to exclude an aneurysm of the aorta, a *bougie* was passed from time to time. The stricture was always present. The upper limit of the obstruction in this patient was, by means of the bougie, ascertained to be, on every occasion, eleven and a half inches from his incisor teeth,—that is, about four inches above the cardiac end of the stomach.* In spasm of the Œsophagus the point where the obstruction commences is practically never constant. As I have said, he lost weight considerably at first, but since he has had a bougie passed he has not lost flesh at the same rate, and during the last fortnight his weight has not varied. Patients with malignant stricture may improve slightly; first, because under treatment more food is artificially introduced into the stomach, and secondly, because ulceration or the frequent passage of a bougie may carry away small pieces of the growth, and by thus slightly increasing the lumen enable swallowing to be performed. If, therefore, there is temporary improvement it must not be assumed that the case is one of spasmodic and not malignant stricture. Malignant disease of the Œsophagus kills by direct extension rather than by secondary growths. This is only to be expected when we remember that the growth most frequently found in this situation is a squamous epithelioma.

* By the middle of March the upper limit of the obstruction had extended to a point ten and a half inches from the incisor teeth.

MEETING OF THE SOCIETY OF ANÆSTHETISTS,

At 20, Hanover Square, March 17th, 1898,

The President, Dr. DUDLEY BUXTON,
in the Chair.

Mr. A. COLEMAN read the following paper,
entitled

On a Method of Administering Anæsthetics through the Nose.

GENTLEMEN,—By the kind permission of your President I have the pleasure of bringing under the notice of this Society an apparatus for administering anæsthetics through the nose, with the object of prolonging unconsciousness during operations on the mouth. Before, however, describing the apparatus allow me to take what the racing fraternity would term a short preliminary canter. The subject of anæsthetics was to myself always one of considerable attraction, and in the department of surgery to which I devoted most of my life, naturally, their employment for operations on the mouth engaged my special attention. At the period I commenced practice the anæsthetic almost universally employed, in this country, at least, was chloroform. Ether was very rarely used, for the reason that we did not know how to administer it. I remember Mr. Stanley of St. Bartholomew's, after a recent death from chloroform, requesting the anæsthetist to the hospital to give ether to one of his patients. It was applied on a large conical sponge, with the result that at the end of half an hour the patient was somewhat excited, and the process had to be completed with chloroform. It might seem to most gentlemen present that in chloroform (putting aside the element of danger) we had an anæsthetic under which almost any dental operation could be undertaken and completed at a single administration. But such was not the case in the days—the forties and the fifties—of which I am speaking. The dental practitioners of that period were accustomed to operate very deliberately and slowly, and no doubt with the smaller risk of fracturing the teeth, of injuring, beyond absolute necessity, surrounding structures, and of causing more than the minimum amount of pain, and therefore it was necessary

occasionally to repeat the administration several times. Sir Benjamin Ward (then Dr.) Richardson was the first to try in an operation on the mouth the insertion of a tube, connected with a chloroform inhaler, into one nostril, but he probably obtained but little advantage from this method. Soon after seeing an account of Dr. Richardson's experiment I had an instrument constructed where a thin metallic tube went into each nostril, but with this I obtained no very great advantage until I connected with the inhaler—a Snow's portable one—an air syringe which enabled me to force the mixture of air and chloroform vapour into the pharynx. By this arrangement anæsthesia, with the mouth open, could be kept up indefinitely. Still, it was difficult at times to avoid giving an overdose, and though I never heard of any fatal catastrophe occurring under its employment, I myself had, and I know others also had, cases which gave them some anxiety. But the necessity for a means of prolonging the anæsthesia became vastly more important when nitrous oxide came into use, and here it was that our friends of now quite a past generation were sadly perplexed. They spoke of the agent in no very complimentary terms, and their patients were in much the same condition of mind as the Irish gentleman who described his experiences of a ride in a Sedan chair. "If it had not been for the honour of the thing he would rather have walked." The unfortunate gentleman had been put into one without bottom or seat!

I had long found that by forcing air mixed with ether into the nostrils after the removal of the face-piece when gas had been administered, some prolongation of the anæsthesia was obtained, but the slight delay in changing the apparatus, combined also with interference with the operator, were drawbacks to its employment. To avoid these I have constructed the apparatus now before you. It consists of a nose-piece made to loosely cover the nose and fit accurately to its base, and which is connected with a very flat tube also adapted to fit accurately over the forehead. To the latter is attached a piece of stout rubber tubing having at its further extremity a very lightly constructed two-way stopcock which is connected with the ordinary gas-bag, but between the two is placed a valve which opens towards the nose. The gas-bag is connected by a long tube to the gas-bottle. When using the apparatus it is well, as

Dr. Hewitt, who has very kindly assisted me in some experiments, suggests, to instruct the patient to breathe in at the nose and out at the mouth for a few times, and then to adapt the nose-piece, the same form of breathing being continued. An air-padded face-piece, rather larger than is ordinarily employed, and having only the outlet valve, is placed over the nose-piece and mouth and the gas turned on. When the patient is fully narcotised the face-piece is removed and the operation commenced, whilst sufficient gas is admitted into the bag to slightly distend it beyond its normal capacity. My friend Mr. Paterson, one of the anæsthetists at St. Bartholomew's Hospital, has kindly employed my apparatus, and has favoured me with the following results of his trials with it. He says, "I have used your apparatus in fifteen dental cases, the following being the durations of anæsthesia :

	Min.	Sec.		Min.	Sec.
Case No. 1 ...	4	0	Case No. 9 ...	3	40
" 2 ...	2	50	" 10 ...	1	20
" 3 ...	4	0	" 11 ...	0	50
" 4 ...	1	50	" 12 ...	3	40
" 5 ...	1	40	" 13 ...	5	0
" 6 ...	1	30	" 14 ...	3	10
" 7 ...	1	10	" 15 ...	1	10
" 8 ...	1	30			

In all the cases the administration could, I believe, have been continued for a longer period had it been required. The longest case was exactly five minutes, and during this time 13 teeth were removed, several of them difficult extractions."

Gas, ether, and air. In employing the apparatus for this combination, a vessel constructed to contain ether immersed in a larger vessel filled with warm water is connected between the gas-bag and the gas-bottle, and is likewise connected with a Fletcher's foot-blower. The ether-containing vessel is so constructed that by turning a two-way stop cock both the gas from the bottle or air pumped from the bellows can be passed under the surface of the ether on their passage to the gas-bag. The process is conducted in the first place precisely as with gas alone until the patient is nearly unconscious, when the gas is diverted through the ether. When complete narcosis has arrived the gas is turned off and air pumped into the bag, when of course after a few inspirations gas and air only will be inhaled. When I recently com-

menced trying the apparatus I have described I was not aware such good results could be obtained with gas alone, and thought that most probably this gas and ether arrangement would be that generally selected for use. I think, however, that most anæsthetists will, for dental operations, prefer employing the gas alone if anything like the same results can be obtained.

Chloroform inhalation.—Whilst thinking over this method of giving gas and ether it occurred to me that by a modification of the instrument it might be made capable of giving chloroform on the Clover plan, *i. e.* mixed with air in absolute definite proportion without the cumbrous appliance he employed. My idea is that air may be propelled by the bellows, and in quantities that may be regulated over the surface of chloroform into an ordinary gas-bag. The proportion of chloroform vapour taken up will of course be in direct relation to the amount of air passed over its surface in a given time, corrections being made for atmospheric temperature, and, indeed, possibly for pressure also. It will take a large number of observations before a dial such as is shown in the diagram can be graduated to indicate the exact percentage of chloroform contained in the gas-bag, but I have no fear ultimately of my not being able to accomplish this. The extremely valuable and interesting paper read at the last meeting of your Society by Professor Waller again directs our attention to the vast importance of giving chloroform and air in definite proportions as was carried out on the Clover principle.

(To be continued.)

Treatment of Alcoholic Meningitis. By Dr. Chas. L. Dana ('Med. Rec.,' 1897).—Treatment is to be instituted from the beginning of the disorder. If any signs of debauch are left, wash out the stomach and give a purge. Feed the patient liberally with hot milk given every two hours. Avoid whisky as a stimulant, but give strychn. gr. 1—10 every two hours. Apply ice cap to the head, and blisters to the nape of the neck. If the patient becomes comatose, tap the spinal cord. The author cites fifteen cases in which amelioration of the symptoms occurred after tapping.—*Post-Graduate*, May, 1898.

THE SURGICAL AFFECTIONS OF THE STOMACH, AND THEIR TREATMENT.

A Course of Lectures delivered at University College during February, 1898, by

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No. VI.

GROUP III.—PREVENTIVE OPERATIONS.

GENTLEMEN,—We come now to the third and last group of surgical procedures for the remedy of conditions in the stomach which are held by many to require operation. These I have called the *preventive* operations rather to emphasise the fact that such *may* be undertaken to prevent ultimately dangerous conditions arising in the stomach, as well as for remedying them when they are present. But of course when you come to consider these operations you will notice that some of them, at all events, might be considered remedial quite as well as preventive; but for the sake of emphasis I have put them, as it were, in a group by themselves. To refresh your memories I may put the table showing the grouping of the operations before you again (Table XI). Under the heading of preven-

TABLE XI.

Operations for Non-malignant Disease of the Stomach.

Group I.—Emergency Operations :

- (a) Gastrorrhaphy for Perforation.
- (b) Gastrotomy for Hæmorrhage.

Group II.—Remedial Operations :

- (a) Divulsion for Pyloric Stenosis.
- (b) Pyloroplasty for Pyloric Stenosis.
- (c) Gastroplasty for Hour-glass Stomach.
- (d) Gastro-anastomosis for Hour-glass Stomach.

- (e) Gastroplicatio for Dilated Stomach.
- (f) Gastrolisis for Adhesions.

Group III.—Preventive Operations :

- (a) Pyloroplasty for Pyloric Stenosis.
- (b) Gastro-enterostomy for all the above.

tive we put the two gastro-enterostomies, anterior and posterior (Figs. 5 and 6), and one might add to these pyloroplasty and gastroplasty (Figs. 7, 8, 9, and 10). But there is a tendency to rely more upon the two gastro-enterostomies for the

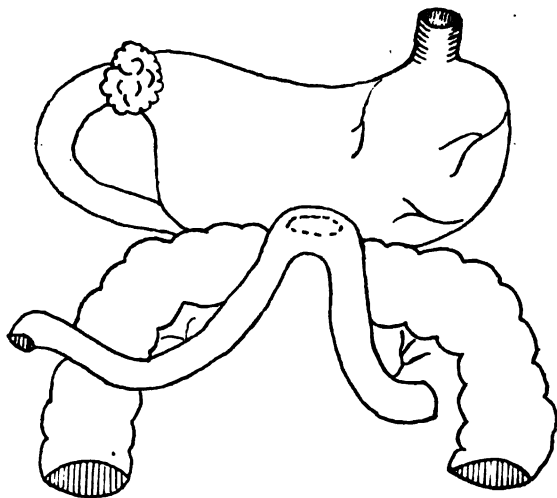


Fig. 5.—Woelfler's anterior gastro-enterostomy.

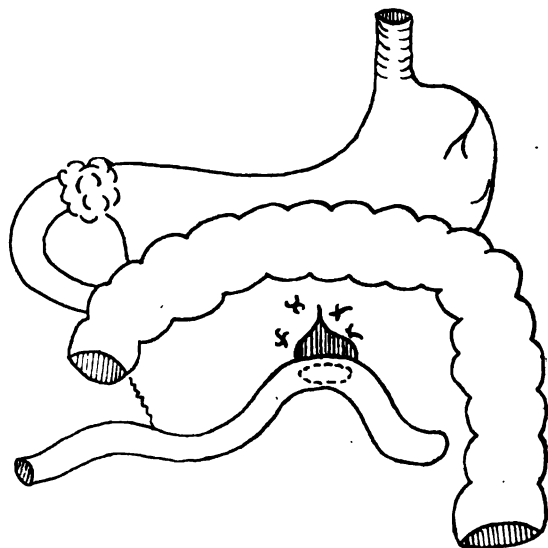


Fig. 6.—v. Hacker's posterior gastro-enterostomy.

remedying of conditions to which we shall allude, than upon pyloroplasty and gastroplasty.

Now no one studying the figures referred to in our previous lectures, and having had any experience in the surgical treatment of the sequelæ of gastric ulceration, can fail to ask himself the

question sooner or later, whether in cases where medical treatment is obviously failing to cure the morbid condition of the stomach, surgery cannot interpose to arrest the disease and prevent those untoward consequences which follow in such a considerable number of cases, as we have already seen.

This question, occurring of late to physicians and surgeons, has prompted surgical measures in many cases for chronic gastric disease, *i. e.* those I have called prophylactic or preventive operations; their aim being to forestall those various conditions

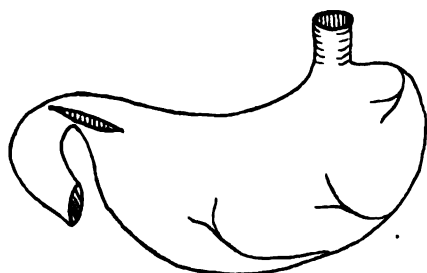


Fig. 7.—v. Heineke's pyloroplasty. 1st stage.

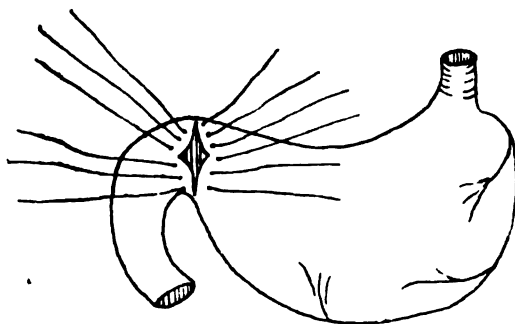


Fig. 8.—Pyloroplasty. 2nd stage.

which frequently supervene upon the intractable gastric disturbance. The line of reasoning adopted by those who answer the latter part of our question in the affirmative is this:—a certain limited number of patients afflicted with chronic gastric ulcer of an intractable kind certainly run serious risks of perforation and hæmorrhage in the first place, while others are condemned to prolonged suffering from the pyloric stenosis and dilatation, and several more remote dangers depending on the latter. A third group, although not in any danger perhaps of immediate or ultimate death from the ulcer, are in constant discomfort, if not

suffering, from a disorder which has been unrelieved by medical treatment, and have to lead a life more or less of chronic invalids, with a prospect of aggravation of the affection rather than relief.

Inquiring into the ultimate course of all these disorders, many clinicians believe that they can be traced to functional or structural changes in the pylorus in almost every case, due to what may be called for shortness imperfect digestion. Their view of the mechanism of digestion, broadly stated, is this:—"that the introduction of food into the stomach calls forth, normally, a contraction of the pyloric sphincter, and that the latter only relaxes

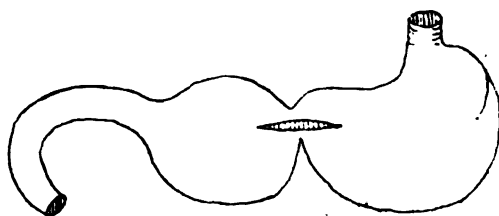


Fig. 9.—Gastroplasty. 1st stage.

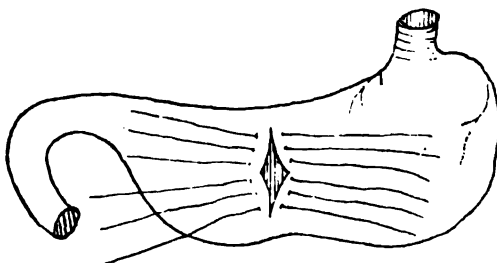


Fig. 10.—Gastroplasty. 2nd stage.

and permits the escape of the gastric contents into the duodenum when the meal has been reduced by the action of the gastric juice to a liquid or semi-liquid form." The pylorus stands sentry as it were over the exit from the stomach, and will let nothing pass through into the duodenum which has not been reduced to a condition suitable for absorption in the small intestine. As you have been told elsewhere, this reduction of the food in the stomach takes place normally in from six to seven hours.

"Every pathological condition which hinders or delays this metamorphosis leads to contraction or spasm of the pylorus, and consequent delay in the emptying of the stomach. The first and most injurious result of this state of things is fermenta-

tion, which never fails to occur when food is retained too long in the gastric cavity. A few years ago it was believed that the free hydrochloric acid of the gastric juices prevented decomposition, and this was supported by experiments which showed the antiseptic action of a watery solution of hydrochloric acid in various stages of concentration."

"But the investigations of many observers have since shown that the action of hydrochloric acid in the gastric juice is very different from the watery solution of that acid employed outside the stomach. According to Strauss, it does not here exert this action at all in the strength given as present in the gastric juice. Even hyperchlorhydria, as an excess of hydrochloric acid in the stomach has come to be called, does not prevent decomposition or fermentation; it is rather one of the first and most frequent causes of the same. It delays the digestion of starchy food, and this calls forth a contraction of the pylorus and a consequent retention of the stomach contents."

"The same fermentation takes place when digestion is delayed, either by hypo- or hyperchlorhydria in consequence of any organic disease of the organ."

"We know that fermentation also takes place in the normal stomach, for the organ contains, as a rule, a great number of bacteria of various kinds, and, moreover, this fermentation occurs in the earlier hours of digestion, but it only develops slowly, and then leads to no ill results, when the contents of the organ pass readily into the bowel, where these bacteria exercise a beneficial influence upon the digestion, as was shown by Pasteur and others." "Many observers have noticed, too, that patients showing entire or almost entire absence of hydrochloric acid (anachlorhydria) nevertheless preserve their health when the stomach empties itself regularly."

I have thought well to give you these views in Professor Carle's own words, as one of the most ardent exponents of the newer views as to the surgery of this organ; and much of what follows is merely a brief digest of his expressed opinions, which are supported by many other observers, and among them by Doyen of Paris, and in a modified way by Mikulicz, who does not go quite as far as they do.

"But even with hydrochlorhydria patients suffer

little so long as the muscular tissue of the stomach is sufficiently powerful to overcome the cramp of the pylorus. In such cases if alkaline fluids are used they neutralise the excessive acids and control the irritants which affect the pylorus, so that the latter relaxes and allows the fluid to pass." "The conclusion drawn from this premiss is important, says Carle, namely, that the origin of every disturbance of the stomach in those suffering from so-called indigestion, whether it lead to excess or deficiency of hydrochloric acid, is undue retention and stagnation of the food in the organ."

It seems a natural conclusion to draw from the premises given, and if the premiss is correct the conclusion is probably correct too. This retention of food, however, is not always due to simple spasm of the pylorus. Besides obstruction by malignant growths, by foreign bodies such as gall-stones, and the pressure of tumours from without, obstruction may be brought about in several ways. (Apropos of gall-stones I may mention that in one of Mikulicz's cases, when he began to do a pyloroplasty he came upon a hard substance, which to his amazement was jammed in the orifice of the pylorus. This he removed, and found it to be an enormous biliary calculus.) Obstruction may be caused (1) first by genuine fibrous stenosis of the pylorus, due either to ulcer or pyloritis, or inflammation reaching it from without. (2) It may be produced by spasm due to ulcers or fissures of the mucous membrane of the pylorus, just as in a case of fissure of the anus we have contraction of the sphincter, or a fissure at the corner of the mouth or of the eyelid. Or, again, (3) it may be brought about by an alteration of secretion; and thus retention of food in the stomach may be brought about by primary atony of the stomach, which of course would lead to accumulation of food in the part.

The relative frequency of these affections may be judged, to a certain extent, by an analysis of forty-four cases operated on by Professor Carle, among which fourteen were pyloroplasties, three divulsions, and twenty-seven gastro-enterostomies. And I might point out to you that an examination of a number of cases *in vivo* ought to lead to more correct conclusions than those which could be based upon a clinical examination and diagnosis in the ordinary way. The diagnosis in many cases may be wrong, and you will see that clinical examination, backed up by an occasional autopsy,

is not so likely to be correct as the observation of an unbroken series of cases in which there has been what some people call an autopsy *in vivo*. Now these forty-four cases arrange themselves thus:

Cicatricial stenosis was found in	30 cases.
Simple spasm	" " 9 "
Atony of the stomach	" " 3 "
Duodenal stenosis	" " 2 "
	—
	44

In thirty-three of these the condition was due to cicatricial stenosis, in nine to simple spasm of the stomach, in three to atony of the stomach, and in two to duodenal stenosis. That is as the conditions were found at the time of operation. Among those who were found to be suffering from cicatricial stenosis, the cause in four cases (and this is exceedingly interesting) was the action of caustic acids,—carbolic, hydrochloric, nitric, and sulphuric respectively, swallowed in attempts at suicide. These cases came under Carle's treatment with marked evidences of pyloric stenosis, were operated on, and the conditions carefully studied at the time. In these four patients the contraction was rapid—a very interesting point: by rapid I mean it took place in one and a half months, two months, four months, and six months respectively, and the contraction was very severe. You know that contraction following on chronic ulcer of the stomach is very slow, and may occupy years. Now this contraction was most marked about the pyloric end of the stomach, as might have been expected when we consider the anatomical position of the organ in the contracted state. Those anatomists who have gone most carefully into the matter tell us that the pylorus is the deepest part of the gastric cavity when the patient is in the vertical position, if the stomach is empty and contracted. Of course when it is full of food the great curve descends and drags down the lesser curvature. Therefore the pylorus in the empty stomach being the deepest part, acid thrown in from above would reach it very quickly, the pylorus would contract on it, and the acid would be held in the pyloric antrum rather than in the deeper curve, and would exert most of its influence on the distal end of the organ. In one of the cases, however, the middle of the organ was the part most affected by the acid, and an hour-glass contraction was the result. In two of the four cica-

tricial cases due to caustics, a great deficiency of hydrochloric acid was noticed, and was explained by Carle as probably owing to atrophy of the mucous membrane and its glands, following on the inflammation set up by the acid. It is a reasonable explanation, for chronic inflammation destroys the secreting surfaces. In all the remaining case hyperchlorhydria was present, namely, in all the other cases of spasmodic or fibrous stricture of the pylorus, as proved by the washings of the stomach and analysis afterwards. And I may say that this group of cases gives us the largest material I am aware of for the study of the changes taking place in the stomach during digestion and after these operations, because Dr. Fantino, Professor Carle's assistant, went to the greatest trouble in washing out these stomachs before and after operation at stated intervals, and analysing the washings with a view to ascertain what was the state of digestion and the amount of hydrochloric acid, and also whether lactic acid was present or not. In the course of that study he followed the course of the digestion by what are called the test meals devised by Ewald, Leube, and Boas, with the view of bringing out into prominence the actual condition of the stomach secretions. Therefore we have here a very accurate estimate of what takes place in the stomach.

The next point which comes out from the study of these forty-four cases operated on is that chronic ulcers of the pylorus are frequently present without any special or very definite symptoms. Bleeding, which is generally supposed by writers on the subject to occur in one-third to one-fourth of all cases of gastric ulcer, was found to have been present in only three of the series. When we remember that these cases were operated on and the actual state of the stomach studied *in vivo*, this point is of importance as giving us more accurate data than those which are derived from many clinical observations. It shows how untrustworthy this symptom is as a means of determining the presence or absence of gastric ulcer. The same fact came out in my own nine cases operated on; bleeding was not a prominent symptom in those cases. As an illustration of the class of cases operated on by Carle, let me give you a brief extract of some of them. Nine stomachs when exposed showed such scarring when examined with the eye and hand as to leave no doubt in regard

to previous ulceration. In No. 9 a fibrous cord three or four centimetres thick ran from the pylorus to the right, and united the parietal peritoneum with the anterior edge of the liver. The serous surface of the pylorus was red and covered by old membranes, and adherent to the pancreas. The pylorus was thickened and its lumen reduced to the size of a goose-quill, and there were distinct scars on the mucous surface. In No. 20 a stellate and very thick hard scar was seen in the duodenum, and caused stenosis. In No. 21 extreme stenosis was found, and the pylorus formed a very hard irregular ring surrounded by indurated glands. The second part of the duodenum was much dilated, so that it was easy to bring it into contact with the greater curvature of the stomach, and gastro-duodenostomy was therefore performed successfully. In this figure (Fig. 6a) I have

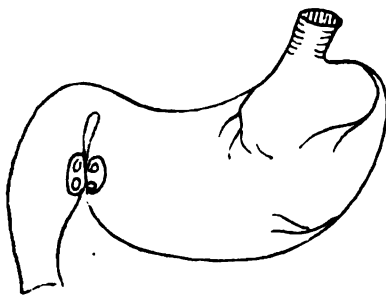


Fig. 6a.—Gastro-duodenostomy.

represented the dilated duodenum and stomach almost in contact with it, and it only remained for Professor Carle to put in a Murphy's button between the two and unite the dilated duodenum at this spot (a) with the dilated stomach, thus making a new pylorus. In No. 22 the pylorus was contracted by white glistening stellate scars, especially on the upper and anterior aspect, and would hardly admit the tip of the little finger. No. 25 showed the pylorus in some parts almost a centimetre thick, fibrous and irregular, with firm external adhesions. In No. 28 there was a hard irregular mass with adhesions in the pylorus. No. 27 exhibited an ulcer with stellate scars, slight stenosis, and peripyloric adhesions. No. 29, a hard thick narrow pylorus with white stellate scarring. In none of these eight cases had there been any hæmorrhage or any other perfectly characteristic symptom of ulcer. Besides, he operated on another case in which there had been

no bleeding, but with such symptoms that carcinoma was diagnosed beforehand. That case was as follows:—No. 19, a tumour the size of a hen's egg involved the pylorus and lesser curvature. It had infiltrated the gastro-hepatic omentum, and adhered to the liver. On opening the stomach a crater-like ulcer was found which had destroyed all three coats of the organ, and penetrated the liver for more than a centimetre. There never had been bleeding. It was proved by microscopical examination to be non-malignant and purely inflammatory. Then followed three cases in which hæmorrhage was present, that hæmorrhage having chiefly determined the operation. No. 18, gastro-enterostomy. On the anterior wall of the stomach near the pylorus was a stellate, depressed, hard scar, the whole pyloric region being taken up by thickening with external adhesions. Pyloric opening very small. No. 16, divulsion. Diagnosis cancer, stellate depressed scar on the posterior surface of the stomach, four or five centimetres from pylorus; latter adherent to pancreas. No. 40, hæmorrhage. Pylorus very narrow from peripyloritis.

There were twelve other cases in which direct examination of the mucous membrane was not possible, therefore it could not be proved whether ulcer existed, though external appearances rendered it very probable. In four of these twelve cases the pylorus was not very much narrowed, and the stenosis was due to faulty position caused by external adhesions. I ask you to carefully note that. Adhesions involved the gall-bladder four times; the great omentum, gall-bladder, and pancreas once. Adhesions from pure peripyloritis were found nineteen times in the forty-four cases, that is 43 per cent. When we add to these the cases of inflammatory fibrous stricture of the pylorus, the percentage rises to 63. In Nos. 26 and 28 the cause of obstruction to evacuation of the stomach was purely mechanical, either definite intrinsic fibrous shrinking or contraction of external adhesions. But another class of case was also noticed in this series, in which the pylorus was not organically narrowed, and was free from adhesions, and yet the patients had been reduced to a state of great weakness by constant vomiting and violent gastralgia, the organ being overlaid and dilated, suggesting pyloric stenosis, although none was present. This was not due to atony of the stomach,

for the latter in each case was capable of violent contraction, sufficient to eject the contents at least three feet from the patient's mouth during vomiting. And in all except two of these cases the muscle substance of the stomach was found strikingly hypertrophied. Now these are the cases which are put down to simple cramp or spasm of the pylorus. But mark, again, there was no organic change found in the pylorus, and the stomach was not atrophied. They all showed gastric dilatation, and after pyloroplasty were relieved by the larger outlet provided. In seven of these cases there were no coarse anatomical lesions whatever in the pylorus. In two there were slight physical changes. In five of the seven cases there was continuous well-marked hyperchlorhydria, and in the remaining two there was some excess of hydrochloric acid, so that it was in excess practically in all. I think these observations are as accurate as any of the kind can be, as they were very carefully carried out. They are on the same lines as the work of others, but no one seems to have been so systematic in the matter of daily washings. In fact, I wonder the patients endured it, but they appear to have done so. These cases of spasm of the pylorus are believed by those who have seen them to be due to the irritation of starchy food which is undigested in the presence of free hydrochloric acid. It may also be produced by the stimulus of slight scratches and fissures in the mucous membrane. It seems likely from all this that a vicious circle is established in some dyspeptic people. Improper food calls for increased secretion of the hydrochloric acid, which may retard the digestion of the food. The pylorus refuses to allow this to pass undigested, and contracts spasmodically. This leads to decomposition of the food and the further aggravation of the spasm.

In the series of cases I have been mentioning to you there were three of true gastric atony. These were not cured by pyloroplasty, which had been done in order to relieve the stomach, which in each case was much dilated. One was relieved subsequently by posterior gastro-enterostomy, and the reason given is this, that in gastric atony the stomach would not be so completely evacuated by enlargement of the pyloric opening, which is more or less above and in front, as it would be by posterior gastro-enterostomy, where gravitation would tend to empty the stomach rapidly, and

therefore the organ would have a greater chance of recovering itself.

The cases of non-malignant stenosis are given in the table I put before you, taken from the list of forty-four cases already dealt with (Table XXVI).

Table XXVI.

Causes of Non-malignant Pyloric Stenosis found in Forty-four Cases operated on by Carle.

Cicatrices from caustics	4*
Ulcers with hæmorrhage	3
Ulcers without hæmorrhage	9
External adhesions	13
Adhesions with spasm	9
Chronic pyloritis	3
Primary gastric atony	3
Duodenal stenosis	2†
	—
	46

* One placed with spasmodic cases.

† One combined with pyloric stenosis.

Out of all these cases there were, as you see, cicatrices from caustics in four, ulceration with hæmorrhage in three, ulcers without hæmorrhage in nine, external adhesions in thirteen, adhesions with spasm in nine, chronic pyloritis in three, primary gastric atony in three, duodenal stenosis in two. This makes forty-six, which is accounted for by one of the cases of cicatrices being quoted again as one of spasm, and one of the duodenal cases as one of contraction of pylorus. I will just note in passing that primary atony would not naturally lead to stenosis of the pylorus, but when accompanied by decomposition of the food consequent on it, it would tend to cause spasm of the sphincter.

Now as to the operative treatment of this class of cases. In all the work done by Carle, from whose figures I am quoting as the most recent, he has limited himself to the operative treatment only of the severest cases, in which internal remedies had had a full trial, often for years, without benefit, and in which the patients had fallen into a state of extreme misery: there was emaciation the consequence of obstruction, pain, sleeplessness, distension, abnormal fermentation, and absorption of septic products, and these symptoms were the motive for the operation. Remember that the stomach as a rule is not specially designed for absorption of food, but if its surface be raw or abraded it can after a while absorb septic products. Some writers even put down a definite form of

tetanus as produced in this way. Twice the operation was done on account of severe hæmorrhage, both patients recovering, one after gastro-enterostomy and the other after divulsion.

The forty-four cases alluded to were treated variously, twenty-seven by gastro-enterostomy, three by divulsion, and fourteen by pyloroplasty. The aim of gastro-enterostomy in such cases is (1) to relieve all obstruction, both of the stomach and in the upper coils of bowel; (2) to make regurgitation of the bile into the stomach difficult. Both anterior and posterior gastro-enterostomy were performed in different cases. The results obtained in each case were so different that Carle strongly advocates the posterior method. The reasons given for this are, briefly:—although the anterior method can be performed with less manipulation in the abdomen, and consequently more rapidly, it does not leave so perfect a physiological condition behind as does the posterior anastomosis. There is the dragging of the small intestine on the large one, and dragging of the large on the small, and the opening being anterior, the duodenal contents as they flow down have a tendency to regurgitate into the stomach. Moreover there is kinking of the bowel almost invariably opposite the aperture made, and instead of the stomach contents going into the efferent portion they are forced back into the duodenum. Von Hacker's method, on the other hand, by placing the opening behind helps to empty the stomach by gravity. Here, again, there is no kinking, and no parallelism between the afferent and efferent portions; and although there is regurgitation of bile into the stomach, in that case it very readily gets out again, and it has never been vomited in his cases.

Now as to the immediate results of these gastro-enterostomies, I shall deal with them very briefly. In nearly all, if not all, it was found that regurgitation of bile into the stomach took place, and that whether the latter was empty or contained food. This was tested by the syphon tube. This regurgitation of bile was almost always more abundant after the anterior operation than after the posterior, but it occurred also in all the posterior gastro-enterostomies, the quantity drawn off sometimes being as much as 800 cubic centimetres. But in posterior gastro-enterostomy this bile never seemed to cause vomiting, whereas it always did so where the anterior operation had been chosen. I have

seen this bilious regurgitation in some of my own cases, which were all done by the anterior method. It is true it did not produce any ill effects in them, but still it was there. Again, the reflux of bile into the stomach became less and less with time, probably owing to the formation of a sphincter. On the whole, in the case of the posterior operation this bilious regurgitation led to no evil results.

In considering the ultimate results of gastro-enterostomy I ask you to just glance at them under the following heads:—(1) As to the change in the motor activity of the stomach,—that is, whether it recovers its muscular power or not. (2) As to the presence or absence of sphincter action in the new opening. (3) As to the capacity of the stomach, *i. e.* whether it recovers its normal size. (4) As to the secretion of hydrochloric acid.

(1) Now, as regards the first point, the operation always brings considerable improvement in motor activity, but *perfect* recovery is rare, at all events for some considerable time. But after a few months the stomach is emptied sooner after this operation than in the normal physiological condition.

(2) As to the presence or absence of a sphincter, the new enteric pylorus was found to retain the food in the stomach adequately, and gave evidence after a time of the formation of a sphincter. I cannot go into the details of proof for that conclusion, but it was shown to be so.

(3) After all but one of these operations the capacity of the stomach was found to diminish. In one case recovery from dilatation was so marked that the lower border of the organ, which before the operation reached to within five centimetres of the pubes, was, five months later, four or five centimetres above the umbilicus.

(4) As to the secretion of hydrochloric acid, Carle draws a distinction between those cases showing hyperchlorhydria and anachlorhydria before operation. This is a matter which I think it is important to bear in mind. First as to hyperchlorhydria, if present before surgical treatment it was found to cease as soon as the patient recovered from the operation. The hydrochloric acid varied ultimately in amount from 0 to 1·5,—that is to say, it was considerably less than usual. As to hypochlorhydria and anachlorhydria when present—that is to say, the reduced amount or

absence of hydrochloric acid,—these cases were not affected by the operation in this respect. This is probably explained by the changes which had taken place in the mucous membrane, such as atrophy of the gastric glands. Of course, if these were destroyed hydrochloric acid would not be present, and if disease has lasted a long time in the stomach it is extremely likely that these gastric glands would be destroyed. But although there was no increase of hydrochloric acid in these cases after operation the patients were benefited by the procedure.

I now take the ultimate results of pyloroplasty and divulsion taken together, contrasted with those just given of gastro-enterostomy. In thirteen cases of pyloroplasty operated on between 1889 and 1893 the period of observation was from three to seven years, so that we may say there was a good opportunity of estimating what benefit these patients had derived from the operation. It may be said generally that they were perfectly relieved, and could enjoy any kind of food. On the whole, the results of pyloroplasty and divulsion in regard to the functions of the stomach corresponded to those of gastro-enterostomy, except that there was no regurgitation of bile into the stomach in these first two operations. But the motor activity of the stomach was not so soon regained after pyloroplasty as after gastro-enterostomy, especially when the posterior method was employed. That, I think, is perhaps easily explained. The escape of the fluid into the posterior opening relieved the stomach more rapidly than when the pylorus was simply enlarged. Again, pyloroplasty had little or no effect on the stagnation due to primary atony of the stomach, in contrast to the effect upon the same condition by posterior gastro-enterostomy. In one case the latter operation relieved this condition, whereas pyloroplasty, which had been previously performed on the patient, had not done so.

Taking all these points into consideration, together with the fact that the mortality by gastro-enterostomy seems a little lower than that of pyloroplasty, it appears likely that in future posterior gastro-enterostomy will be preferred to pyloroplasty where operations are to be done for these conditions. However, Mikulicz has come to a different conclusion, namely, that pyloroplasty is the better of the two operations. He has put

forth arguments in favour of that view. Perhaps there is not much to choose between them at the present time, and you must remember of course that we are dealing with one of the newer branches of surgery, and one upon which we are still in want of evidence, although it is being furnished daily in ever-increasing ratio. We cannot, therefore, come to any final conclusions yet upon the point from the sparse information before us.

We have now come to the end of these lectures on the subject of gastric surgery. You will probably remember what I said about the latter at the beginning, that I should endeavour simply to give you a review of the subject, and not advocate this or that line of action. The subject is a very large one, and my difficulty has been rather to choose what *not* to say than what to say,—that is, to select for you what I think would give you a text to think about; and in doing that I have tried most scrupulously to avoid giving you a decided bias one way or another. The conclusion that appears to follow is that certain of these operations on the stomach are justifiable; but the conclusion also to be derived from a study such as this is that the selection of cases will be very difficult indeed, and that we must wait for a considerable time before we can approach a definite decision on the whole matter. I hope, however, that we have at all events had suggested to us in this study some points of interest in regard to the pathology of the stomach, and to the clinical history of its diseases, and some suggestions in regard to the possibilities of surgery for the future in dealing with the latter. He would be a very bold man who should say that these procedures are not going to be taken up warmly hereafter, but are going to be cast aside. All of us here, and those of us who are older especially, are aware of many procedures condemned in the past as absolutely unjustifiable which are now adopted by everybody. I can look back upon many surgical measures which, having been introduced, were strongly denounced at once, and those who had taken them up were pretty generally animadverted upon in the strongest way, and looked upon as next door to criminals. Yet now every second man you meet is doing such operations daily. Therefore it would be a very unwise and, indeed, an unscientific course at once to cast aside these very advanced operations as not being justifiable.

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* Specially reported for The Clinical Journal. Revised by the Author.

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WITH DR. THEODORE WILLIAMS AT THE CENTRAL LONDON SICK ASYLUM,

May 12th, 1898.

LADIES AND GENTLEMEN,—First of all I wish to show you two cases of pleurisy, one slight and the other severe, which are instructive in contrast.

The first patient is a man æt. 30. Five weeks before admission he complained of cough. When he was admitted his temperature went up to 101° in the evening, and was 98° in the morning. It is now 99·2° and 99·7°, so that the fever has practically subsided. It is probably an acute case. One of the early signs of pleurisy is that one side of the chest does not move during respiration as well as the other side. At the present time vocal vibration is scarcely absent. The chief feature is slight dulness, deficient vesicular breathing, and voice-sounds. He says that his breathing now is pretty free. In olden times, and indeed not so very long ago, these patients were not tapped at all, and in many of the cases complete absorption of the fluid took place. The great point is to relieve the tension rather than to take away all the fluid. You require to take away the pressure from the lymphatics and blood-vessels which will be the means of absorbing the fluid, and if you remove the tension, nature will do the rest. Very often it is advisable not to aspirate to the extreme. Another method of treating these cases is to partially starve the patient, and specially to cut down the liquids taken, and that certainly has a good effect.

The next patient is a man of 45. He has had cough sixteen months, and an effusion on the left side has been present some months. On the 13th April he was tapped, and forty ounces of clear fluid came away. Since I saw him the other day he has been tapped again, and on that occasion forty-four ounces of fluid were removed. Therefore the physical signs are altered from those I made out when I last saw him. When I examined

him the whole side was dull, there was absence of breath-sound, and of vocal vibration over the whole of the left side. The heart's impulse could scarcely be felt, but what there was, was to be found in the axilla. Crepitation was to be heard in the upper portion of the left back, in the inter-scapular and supra-scapular regions. The temperature now is 98° ; yesterday it went up to 100° . When he came in it was 99° in the morning, 102° in the afternoon, and 102.6° on several days in the afternoon. The general condition of the patient seems to be fairly good. You see there is present the clubbed fingers, which used to be regarded as characteristic of chronic consumption. In my experience this symptom is more often connected with the chronic pleural effusion, especially with empyema, and with chronic pneumonia, and it is not common in consumption. I beg you to notice that in this case the left side of the chest does not move so freely as the right. I perceive a great improvement in the note since he was tapped. The limitation of the dulness also shows that the diaphragm has been drawn up, and with it the stomach. Over the left back there is a little more breathing to be perceived, and some wheezing sounds can be heard. The lung is close to the chest wall posteriorly, and I fear there is some mischief in the left lung at the top and back. In order to produce collapse of the side, it may be necessary to do something more, such as removing portions of the ribs, and we trust that then the atmospheric pressure will cause the chest wall to fall in gradually. In this way one can assist nature; but, of course, you may get a distorted side, and at any rate you get the chest wall approaching the spine, and a certain amount of connective tissue is thrown out to fill up the void, and a comparative cure results. It is always a trouble when the lung does not rise after aspiration. Sometimes in these cases you find the chest contracting so much as to cause curvature of the spine, the shoulder is considerably lowered, and so on; probably some of you have seen patients of this kind who have the appearance of being lopsided. The curvature is always towards the affected side. You may get the heart-beat in extraordinary positions. Sometimes when one lung does not rise up well, and there is a space to be filled in, the opposite lung will come across the median line and try to fill up the void. In the

present case the stomach has been drawn up to fill up the space.

The third case is an instance of fibroid phthisis, and these cases require careful examination. The patient is a male aged 48. The history is that some years ago he had phthisis. He was in the artillery in 1895, and he appears to have spat blood four years ago. He has had a cough for five years. He again spat blood to the extent of three or four ounces five months ago, and he has been losing flesh for eighteen months. One of his brothers died of phthisis. We now find that on the left side there is considerable flattening. It is one of those cases where inspection goes a long way. On the left side the spaces are contracted below the nipple, and the whole side moves very little on breathing. The heart's apex is three inches below the nipple line. We now try percussion, and find that it is quite dull on the left side, and the line of demarcation is very clear. On the right side there is healthy puerile breathing. In the left axilla, or rather just below it, is the cause of his trouble. At the time the patient spat blood, a cavity formed in his lung, and the cavity itself has been gradually contracting. As a rule, a cavity contracts towards a fixed point. If there is adhesion in any part of the lung, contraction takes places towards that point; if there is no adhesion, contraction occurs towards the root of the lung. In this case the fixed point is in the axilla, consequently towards that adhesion the contracting process is directed. The side has fallen in a little here, and the stomach has been drawn up with the diaphragm. An interesting feature is that the right lung has been drawn across, so that, when you listen over this part, instead of hearing the left lung, you hear the right lung to the left of the middle line. The right lung has become enormously large, to make up for the shrinking of the left lung as far as possible. The signs behind are not pronounced. I saw many patients with contracting cavities during my long connection with the Hospital for Consumption at Brompton, and have seen cases in which the left lung has gone over to the right side, and, *vice versa*, the right lung has gone to the left side. The most striking case I remember was where a cavity formed in the right lung of the patient, and fibrosis took place. The lung was consequently drawn right to the back of the thorax.

We did not see the patient for some time after she left the hospital the first time, but she came back again, and, to our astonishment, the whole front of the chest was hyper-resonant; there was no sign of a cavity, and we could not make it out. We concluded that the right lung had gone across. She went out of the hospital, and came in a year or two afterwards, and this time, to our surprise, there was resonance on the left side, and a certain amount of resonance on the right side; but there were cavernous sounds on the right side under the clavicle. One of my colleagues said it was the old cavity turned up again. The trouble in these cases is not the cough and expectoration; it is the shortness of breath on exertion, and very often they get obstruction of the vessels and dropsy, and many have gone down-hill in this way. This patient eventually died, and we found out that this hypertrophied lung had extended across the median line, and a cavity had formed in that. There was a cavity on the right side, but it was in the left lung. These cases are always of slow progress, and the patients sometimes live a number of years. When they fail it is generally due to obstruction of the vessels and pressure on the circulation. The right side of the heart becomes dilated, and very often the kidneys are affected, and dropsy and albuminuria supervene. I have seen many cases of dropsy and heart disease which have turned out to be cases of phthisis which had been arrested, and after death we found an old cavity and fibrosis of the lung. Another line of development is that the lung becomes hypertrophied and emphysematous, and even bronchitic and asthmatic. There is many a case regarded as chronic asthma and emphysema which is really a case of old tubercle, and there is an enormous amount of emphysema around the old tubercular areas. The disease from which this patient is suffering is what Sir Andrew Clark called fibroid phthisis. He said fibrosis was a disease of itself. Practically speaking, the great mass of fibrosis is due to tubercle. The late Dr. Moxon said that fibrosis was the past tense of tubercle, and there is some truth in the statement; but fibrosis may easily occur after chronic pneumonia, or it may occur as a result of fibroid degeneration of tubercle. In the present case you get a dulness almost as deep as in effusion. A fibroid lung is a solid hard mass, and it is often surrounded by thickened pleura.

REMARKS ON SCARLET FEVER.*

BY

J. W. WASHBOURN, M.D., F.R.C.P.,

Physician to the London Fever Hospital; Assistant
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LADIES AND GENTLEMEN,—It is well from time to time to review our knowledge of a familiar subject, and to see whether our conceptions have been altered by new ideas and fresh lines of thought. With this object I venture to recall to you some points in connection with scarlet fever which appear to me of interest and worthy of consideration.

One would imagine that very little remains to be learnt about such a common and well-defined disease as scarlet fever; but when we inquire into the matter we find that the reverse is true, that our knowledge in many respects is deficient, and that what we do know rests more upon analogy and surmise than upon direct and reliable evidence.

The fundamental problem of the causation of scarlet fever still remains unsolved. The infectivity, the incubation period, the course of the disease, and the protection afforded by a previous attack undoubtedly point to a causal connection with some micro-organism, and yet no micro-organism has hitherto been discovered.

As the disease is not transmissible to animals we have no experimental data to guide us; nevertheless we know something about the *properties* of the virus of scarlet fever, and it is this question to which I would first draw your attention.

We can safely assume that the virus is a micro-organism, but we have nothing whatever to help us in deciding whether it is a bacterium such as the organism causing plague, or a protozoön such as the organism causing malaria.

The principal facts which we know about the virus are the following:

First, the virus is capable of remaining alive for some period outside the body under certain conditions. A number of instances are recorded in which the clothes, bedding, &c., of patients have remained infective for months. The conditions which appear to be necessary for the preser-

* Read before the North London Branch of the British Medical Association on May 4th, 1898.

vation of the vitality of the virus are the absence of sunlight and air.

Secondly, the virus can be transmitted a short distance through the air, and thus infection may occur without actual contact with a patient. Indeed, it is supposed that this is the most frequent mode of conveyance of the disease.

Thirdly, there are grounds for supposing that the virus, like that of typhoid fever, is capable not only of remaining alive, but also of multiplying outside the body. Several epidemics have been traced to the milk supply, and the inference is that the virus has multiplied in this medium. The epidemics I refer to are one described by Dr. Taylor at Penrith, several referred to by Dr. Hart, and one investigated by Messrs. Power and Wynter Blyth at Marylebone. In the latter epidemic, however, it was supposed that the cows supplying the milk were affected with a modified form of scarlet fever. Were this view correct, it would be unnecessary to assume a multiplication of the virus in the milk in order to explain the epidemic; but in the absence of other evidence of the infection of animals with scarlet fever we must hesitate before accepting such a view.

Lastly, the virus of scarlet fever appears to be destroyed by the usual methods of disinfection. I do not know of any instances in which carefully disinfected clothes have conveyed the disease.

So much, then, for the properties of the virus outside the body; let us consider how it behaves within the body.

We shall probably not err in assuming that the virus generally enters the body by means of the fauces. The fact that the disease is frequently contracted by inhalation, the early inflammation of the fauces, and the absence of affection of the lungs, are the points in favour of this view.

But is this the only mode of entrance of the virus? We know that the virus of anthrax may enter the human body in various ways; it may enter through the skin, as in external anthrax, or through the lungs or digestive tract, as in internal anthrax. By analogy we might infer that the virus of scarlet fever does not always enter the body by the usual channel. Let us see whether there are any facts to justify such an inference.

Most writers on scarlet fever refer to epidemics of so-called surgical scarlet fever, by which is meant scarlet fever occurring amongst patients

in surgical wards suffering from wounds of various kinds. The chief characteristic of these epidemics has been their mildness, but this by itself does not go for much. Mild epidemics are frequently met with, not only among surgical patients, but also in schools and other institutions.

I have no doubt that most cases of so-called surgical scarlet fever are ordinary cases of scarlet fever in which the virus has entered the body in the usual way. Nevertheless I believe that *sometimes* the virus enters through wounds, and that the resulting disease is thus modified in severity. My reasons for this supposition are as follows.

The effects produced by pathogenic bacteria depend upon the seat of inoculation; general infection, for example, may result from intraperitoneal inoculation, while subcutaneous inoculation with the same bacterium may produce no result, or at most a local inflammation. Again, smallpox was of a milder type when caused by subcutaneous inoculation than when contracted in the usual way, *i. e.* by inhalation.

Now Dr. Goodall and I have pointed out that patients suffering from burns are more liable to suffer from scarlet fever than other surgical patients. On the assumption that the virus enters through the subcutaneous tissue, the liability could be explained by the extensive surface of body exposed to contagion.

Again, from time to time patients are admitted into the scarlet fever wards of the London Fever Hospital with the following symptoms:—Pyrexia and a typical scarlatiniform rash are present, but there is no inflammation of the fauces. These patients are the subjects of a surgical wound, or more frequently of an open sore in some part of the body, generally the fingers. The disease runs a mild course, and the patients do not contract the disease while in the hospital. I cannot help thinking these are cases of scarlet fever in which the virus has entered through the wound in the skin. There is certainly the possibility that the rash and pyrexia are of a septicæmic nature, but the absence of other signs of septicæmia is opposed to this view.

After the virus has entered the body what does it do? Does it remain localised at the seat of inoculation (in the majority of cases the fauces), or is it distributed all through the blood and organs? Is it a disease like diphtheria, where the

bacilli are chiefly localised in the fauces or larynx, or is it a disease like relapsing fever, where the spirilla are found in large numbers in the blood?

We have, unfortunately, very little to help us in answering this question—of such importance in reference to the conveyance of the disease. If the virus is distributed throughout the blood, the secretions from the skin, kidney, &c., would in all probability convey infection.

There is a general belief that the skin contains the virus, and it has indeed been stated that the patient is most infectious during the stage of desquamation. This latter statement is, however, incorrect, for there is evidence that patients are more infectious during the early stages than at a later period. The view that the virus is contained in the skin may have arisen from the fact that the patient is usually free from infection by the time desquamation has ceased, and a casual connection between the two phenomena has been assumed. But when we consider the matter carefully we find that there is only an indirect connection between the completion of desquamation and the freedom from infection. On the one hand, patients during the stage of desquamation are often quite free from infection. Dr. Priestley, for example, states that 120 children in various stages of desquamation were discharged from the Fever Hospital at Leicester, and that none of these cases conveyed the disease to others. On the other hand, it is certain that patients may remain infectious for some time after desquamation has ceased. In all fever hospitals, so-called "return" cases of scarlet fever occur, by which is meant cases of scarlet fever occurring in a household within a short time after the return of a patient from the hospital. Some of these cases may be attributed to carelessness in disinfecting the patient's clothes at the hospital, and others to the use of infected clothes which had been put aside before the removal of the patient from home. Nevertheless there remain a number of "return" cases which can only be explained by supposing that the patient has continued to be infectious for some time after desquamation has completely ceased. Probably, in these cases, the virus remains in the fauces as it does in diphtheria, in which disease the bacilli may remain even for months after the inflammation has subsided. The practical bearing of these considerations is that we must not look upon the

completion of desquamation as an absolute index of freedom from infection, although it is, perhaps, the best guide we possess.

There is, then, no distinct evidence from the history of infectivity that the virus of scarlet fever is to be found in the skin, and there are no other grounds for believing that the virus is distributed throughout the body. The rash and rheumatism of scarlet fever may be as readily explained by the action of toxines produced by the virus at the seat of inoculation as by the direct action of the virus itself. The later complications, by which I mean the pyrexia, nephritis, and glandular enlargements occurring during convalescence, are comparable to the paralysis following diphtheria, and are probably due to the excretion of toxic substances.

The last question we shall have to consider in reference to the virus of scarlet fever is that of variability in malignancy.

In the case of pathogenic bacteria virulence is the function which perhaps shows most variability. The virulence of a pathogenic bacterium can be exalted by repeated passages through the bodies of susceptible animals; and by appropriate means it can again be diminished.

Let us see what evidence we possess of a similar variability in the malignancy of the virus of scarlet fever.

It is well known that some epidemics are more malignant than others, and that different cases vary greatly in their severity. No doubt this is often due to a difference in the malignancy of the scarlet fever virus, but there are other factors at work which we must bear in mind.

In the first place the predisposition of the patient must be considered. A severe case may be due to an increased predisposition of the patient, and not to an increased malignancy of the virus, and severe epidemics may be due to the disease attacking a large number of predisposed people. You will all be familiar with cases where two individuals have contracted the disease from the same source, the one passing through a mild and the other through a severe attack. I might quote as an example the case of a husband and a wife who contracted scarlet fever at the same time and presumably from the same source, and were admitted into the London Fever Hospital in February of this year. The wife passed through a

mild attack, while the husband died after a short illness of malignant scarlet fever.

In the second place we must remember that a mixed infection with the scarlet fever virus, and with a pathogenic streptococcus, is answerable for the severity of most cases; so that even with a weak scarlet fever virus, and no special predisposition on the part of the patient, the attack of scarlet fever may be fatal.

This question of mixed infections is of exceptional interest, for it plays an important rôle in the pathology of many diseases.

We can distinguish clinically between two distinct types of scarlet fever, the one in which the patient is suffering from the effects of the scarlet fever virus alone, and the other in which he is suffering in addition from a septicæmia caused by a streptococcus.

Most of the fatal cases are due to this double infection, but we now and then meet with fatal cases of pure scarlet fever. Such cases go under the names of toxic or malignant scarlet fever. The main symptoms of this type are those due to a profound intoxication. The temperature is high, the respirations are rapid and laboured, the pulse is quick and feeble, and there are signs of cardiac dilatation. The central nervous system is severely affected, as is shown by delirium and tremor, the rash is vivid and sometimes markedly blotchy. The throat is not severely affected, at most some redness and œdema of the fauces with exudation of dry sticky mucus; and the submaxillary glands are only slightly inflamed. Death usually intervenes in a few days.

A post-mortem examination reveals no changes other than those met with in all cases of pyrexia. No cultivations can be obtained on the ordinary media from the blood or organs, or from the deeper layers of the fauces, and no bacteria can be found on microscopical examination of the tonsils or palate.

I will quote brief notes of two cases of this nature.

Scarlatina Maligna.

1. J. C. D—, male, æt. 36. Admitted into the London Fever Hospital on February 4th, 1898, under my care. Had been ailing since January 29th with indefinite symptoms attributed to influenza. On February 2nd he was better and

went into the country, but returned in the evening feeling ill and cold. On February 3rd he suffered from a sore throat. On February 4th the rash appeared, and he was admitted into the hospital in the afternoon. On admission he looked ill, and was tremulous and dyspnoic. There was a dusky flush over the face, with a yellowish tinge around the mouth, and a bluish tinge on the nose. The scarlatina rash had the usual distribution, and was of a dull livid colour, and slightly blotchy in arrangement. There was moderate congestion and catarrh of the fauces, with slight swelling, but no ulceration. The cervical glands were slightly enlarged. Pulse 140, small and soft; heart somewhat dilated; temp. 104°. Mental condition fairly clear. Frequent retching, but no vomiting. February 5th.—Slept very little during the night, but was not delirious. Said he felt better, but did not look so. Very tremulous and dyspnoic. During the day got worse, and in the afternoon became rambling and incoherent. At 10.15 p.m. he vomited, and immediately died. During the time he was in the hospital the temperature varied between 103° and 105°.

2. W. T. F—, male, æt. 33. Taken ill on July 31st, 1897, with malaise and slight sore throat. On August 1st vomiting and rash. Admitted into the London Fever Hospital on August 4th under my care. On admission there was a dusky appearance about the face, the temperature was 104.6°, the heart-beats 120, with signs of cardiac dilatation, the respirations were 37, and were increased by slight exertion. The rash was profuse and vivid. The fauces were of a bright red colour and slightly œdematous; there was no ulceration. August 5th.—Still dyspnoic, and in much the same condition. August 6th.—The rash beginning to fade, leaving petechial stains on the legs; temp. 105°. The mental condition became confused, and culminated in violent delirium at 6 p.m. August 7th.—Delirious all day, steadily became worse, and died with a temperature of 107.6° just before death.

In neither of these cases was a post-mortem examination made. I have quoted them as they are the most recent ones which have been under my care. In other cases which we have examined no bacteria could be found in the blood or organs.

In the second and more common type of the disease, that of mixed infection, the symptoms are referable to a septicæmia starting in the fauces.

The main feature of this type is severe inflammation of the fauces, from which the name scarlatina anginosa is derived. The tonsils, the soft palate, and the neighbouring parts are considerably swollen, and are covered with pultaceous or even membranous exudation. Extensive ulceration rapidly ensues, and sometimes there is gangrene of the tissues. The glands in the neck are infiltrated, and the inflammation may spread to the cellular tissue, producing the so-called "bull neck." Death, which usually occurs at a later period than in scarlatina maligna, may be due either to a general septicæmia, to some septic complication such as cerebral abscess, or to a general pyæmia. At the post-mortem examination streptococci are found in abundance in the tissues of the fauces and the glands of the neck, and in smaller numbers in the various organs and in the blood.

In this class of case we should expect treatment with antistreptococcic serum to be of benefit, but I cannot claim to have met with any success with this line of treatment.

I will quote brief notes of two cases of this nature.

Scarlatina Anginosa.

1. D. C—, female, æt. 7. Admitted on September 9th, 1897, into the London Fever Hospital under my care. The child was dirty and neglected. No history of the illness could be ascertained, but she had evidently been ill for some time, as the rash had disappeared, and desquamation commenced. She looked ill, and was drowsy and wasted. Pulse 180, temperature 103.2°. The tongue was raw and ulcerated at the edges, the fauces were covered with secretion, both tonsils were swollen and ulcerated, the ulceration extending to the palate. The cellular tissue of the neck was swollen, producing a "bull neck."

The ulceration and inflammation in the neck extended, the temperature ranged between 101° and 104°, the patient gradually got worse, and death occurred on September 13th. During the illness antistreptococcic serum was administered, but without effect.

At the post-mortem examination the ulceration in the throat and the infiltration of the neck were all that could be found; there was no pus formation. Streptococci were obtained on cultivation from the cellular tissue of the neck, the heart blood, spleen, lungs, liver, and kidneys.

2. G. B—, female, æt. 3. On September 21st, 1897, she suffered from headache, vomiting, and sore throat. On September 23rd the rash appeared, and on September 25th swelling of the neck was noticed. Admitted into the London Fever Hospital under my care on October 1st. On admission the child was dirty and neglected, but not wasted. Pulse 140. The tongue was raw, the tonsils swollen and ulcerated, there was a typical "bull neck," and pus was detected on the left side. The rash was fading. On October 2nd a little pus was evacuated by an incision in the neck. On October 3rd the incision was enlarged, but no more pus was found. On October 4th a secondary macular eruption appeared on the limbs. The patient gradually sank, and died on October 11th.

Antistreptococcic serum was administered in this case, but without effect.

Only a limited post-mortem examination was possible. The spleen was removed, and streptococci were obtained from it by cultivation.

The life-history of the streptococcus which produces such disastrous effects is of great interest. In the mouth of all individuals streptococci are constantly to be found. These streptococci when tested on animals appear as a rule to possess no pathogenic properties. Does the scarlet fever virus so lower the resisting power of the body that these otherwise harmless streptococci are capable of invading the tissues, increasing in virulence in their progress? It is an enticing theory, but as yet nothing more. The fact that these septic cases occur more frequently in patients who are neglected and exposed to bad hygienic conditions, rather favours the view that the streptococcus is introduced from without.

There are other mixed infections which may occur in scarlet fever. Dr. Caiger quotes a large number of cases of scarlet fever associated with diphtheria, varicella, measles, and whooping-cough, and a smaller number associated with other diseases, such as erysipelas, enteric fever, and typhus.

The greatest interest, perhaps, attaches to the association of scarlet fever with diphtheria. As a rule, this disease occurs during convalescence from scarlet fever, and there can be no doubt that patients during convalescence are more liable to diphtheria than healthy individuals.

But sometimes the scarlet fever and diphtheria

run concurrently. It is probably in this way that diphtheria becomes introduced into a scarlet fever ward, and that epidemics of post-scarlatinal diphtheria arise. I will mention a case of this nature.

G. C—, æt. 22, female, was admitted into the London Fever Hospital on August 5th, 1897. On August 2nd she suffered from sore throat, on August 3rd from headache and vomiting, and on August 4th the rash appeared. On admission there was a typical scarlet fever rash. Temperature 101·8°. A thin exudation was present on the left tonsil, and the submaxillary glands were enlarged; a bacteriological examination revealed the presence of diphtheria bacilli. Antitoxin was administered, and the patient passed through a mild attack, and was able to leave her bed on August 15th.

On account of the presence of diphtheria bacilli this case was isolated, and no cases of diphtheria occurred in the wards. I believe that a bacteriological examination of the throat of all scarlet fever patients is of value in diminishing the incidence of post-scarlatinal diphtheria in a hospital. At any rate, we have had no post-scarlatinal diphtheria at the London Fever Hospital for the last two years, during which period we have made systematic bacteriological examination of the throats of all the patients.

There are many more points of interest in connection with scarlet fever, but time will not permit me to discuss them now. I will only draw your attention to the occurrence of relapses, and to one point in connection with diagnosis.

Relapses occur in a certain proportion of all cases, and the relapse, like that of enteric fever, is similar to the primary attack in its main features.

Here, for example, is a case. B. E. C—, male, æt. 24, was admitted into the London Fever Hospital on April 20th, 1897, with a typical mild attack of scarlet fever. On May 20th, when desquamation was nearly completed, he was seized with sore throat, and the temperature rose to 100°. On the next day a typical scarlet fever rash appeared, and on May 26th he suffered from scarlatinal rheumatism. A second desquamation occurred.

I draw your attention to the occurrence of relapses because they are often put down to a contraction of the disease in the hospital, the first diagnosis being considered erroneous.

With regard to diagnosis I only want to warn you against mistaking German measles for scarlet fever. At present, on account of the prevalence of German measles, the mistake is frequently made. The usual history is that the medical man first pronounces the case to be one of measles, and then changes his diagnosis to that of scarlet fever. The reason is this. The German measles rash first appears on the face, and the diagnosis of measles is made. It then disappears from the face, and by this time the separate papules have coalesced on the body to form a scarlatiniform eruption. The case is then taken to be scarlet fever, and the patient is sent to the hospital. The mildness of the attack, the slightness of the throat affection, and the history of the rash on the face are the best indications to the diagnosis.

The Administration of Arsenic per Rectum.

—Professor Rénaut, of Lyons, has reported to the Société de Thérapeutique ('Nouveaux Remèdes,' April 24th) the results of his experiences with rectal injections of arsenic. It is administered in solution containing a third of a milligramme in five cubic centimetres. Three injections may be given daily. This treatment may be continued for months without having to be interrupted in consequence of gastric intolerance, and consequently it is admirably adapted to obtain the full effect of arsenic when that drug is indicated as a modifier of nutrition. Should any rectal irritation be caused, the addition of a few drops of laudanum will obviate it. This treatment is highly recommended by the author in tuberculosis, especially in the early stage; in diabetes mellitus, and in exophthalmic goitre.—*New York Medical Journal*, May 14th, 1898.

Takaki and Werner report a case of suppuration of Bartholin's glands in a case of convalescent typhoid fever, twenty-five days after the temperature had reached normal. In the pus a pure culture of the typhoid bacillus was found, and the authors think that their case strengthens the view—now generally accepted—that the typhoid bacillus can produce, unaided, suppurative inflammation; only, however, during convalescence after the system is immune to the general action of the micro-organism. Regarding the portal of infection in their case, they think it may have been local.—*University Med. Mag.*

A CLINICAL LECTURE ON LEUKÆMIA.

Delivered at University College Hospital, Feb. 8th, 1898,

BY

J. ROSE BRADFORD, M.D., F.R.C.P., F.R.S.,

Physician to the Hospital.

GENTLEMEN,—Before discussing the subject of leukæmia, I must say a very few words about the corpuscles of normal blood, simply with reference to leukæmia. As you know, it is customary to say that the normal number of red blood-corpuscles is about five millions to the cubic millimetre, and that within the limits of health it may rise to six millions; and that the normal proportion of white corpuscles varies from 5000 to 10,000 per cubic millimetre,—that is, apart from the influences of diet and so on. From the point of view of leukæmia there is one other set of facts which is very important, and that is the relative proportion of the different kinds of white corpuscles. I am not going into that question at all fully now, but only sufficiently to make clear our subject of leukæmia. You know there are several kinds of white corpuscles, classified by some as different varieties, and regarded by these observers as really different kinds of white corpuscles, while others state that they are merely different stages in the life history of one and the same kind of white corpuscle. There has been great controversy with reference to that, but I do not think we need go into that point, as, although interesting, it has no great bearing on leukæmia.

The white corpuscles may be classified in at least two ways; one according to their staining properties, or rather according to the staining properties of their granules, and the other the classification based on the form and size of the cell, or rather on the form and size of the cell and nucleus. Probably, from a clinical if not from a scientific point of view, the classification based on the size and shape of the cell and of the nucleus is more important, practically, than that based on the staining properties of the granules. Therefore we will divide the different white corpuscles of the blood as follows:—First of all there are what are

known as *lymphocytes*; and we mean by lymphocyte a cell which is characterised by the following properties (I shall not go into the question of the size of the cell, because it undergoes great variation):—(1) the nucleus occupies nearly the whole of the cell; (2) the nucleus has very much the same shape as the cell; and (3) the protoplasm simply exists as a ring round the nucleus. Further, the nucleus is round, and the protoplasm of the cell is clear and hyaline. Hence some have called these lymphocytes, others hyaline cells, owing to the sparsity of granules in the protoplasm. There is another form of lymphocyte in addition to the one just described, which, however, only differs from these in that the cell is somewhat bigger, and the nucleus has not increased to the same extent; otherwise the cell has much the same characteristics as the foregoing. Therefore we talk of small lymphocytes and large lymphocytes, and they are both present in normal blood. Not to complicate matters unduly I may mention that there also are what are called by some people transitional forms. It is an unfortunate name, because you are apt to think that it is a kind of cell which is intermediate between these two, whereas what is meant is one in which the nucleus is no longer round, but has an indentation in it, so that it tends to be lobed. These are the characteristics of the normal lymphocytes, and they form, in normal blood, about 30 per cent. of the total number of white corpuscles; therefore about one in three of the white corpuscles of our own blood consist of that kind of cell.

The next kind of cell which exists in normal blood is one which is generally larger than the small one I have sketched, and smaller than the larger one; and it is characterised by an irregular nucleus. Sometimes the nucleus is so divided that it looks as if there were several; at other times the nucleus looks like a skein of worsted, and this is best described not as a multinuclear cell, because the nucleus is usually single, but as having a multipartite nucleus; that is to say, a nucleus which is very much split up. The protoplasm of this cell is granular; it stains with a number of dyes, and most authorities take the view that it stains preferably with acid dyes, and so it is called oxyphile; but it is not what is known as *the eosinophile cell*. The cell just described is the ordinary white corpuscle of the blood, which

constitutes something between 60 and 70 per cent. (nearly 70 per cent.) of the total white cells of the blood. These two kinds of cells, *i. e.* the lymphocytes and the oxyphile cell with the multipartite nucleus, make up the great bulk of the normal white corpuscles of the blood. There is another cell present in the blood. It is only present to the extent of 2 to 4 per cent. (some people say $\frac{1}{2}$ to 4 per cent.), but 4 per cent. is the maximum, and it is what is known as *the* eosinophile cell. It is characterised not only by the fact that it stains with acid dyes like eosine, but by the fact that the granules stained are very large. I show you a beautiful specimen of this under the microscope. You see that it is coarsely oxyphile, and that the nucleus is partite. Unfortunately, however, these cells may occasionally have a roundish nucleus.

I think that is all I have to say about the normal corpuscles in the blood. The three fundamental points from the point of view of leukæmia are that about one third consist of cells with a round nucleus and with hyaline protoplasm; that two thirds consist of cells with a multipartite nucleus and finely granular protoplasm staining with eosine; and that there is a very small number of cells with coarsely oxyphile granules, which may sometimes have a multipartite and sometimes a round nucleus.

I might say that, with regard to the coarse oxyphile cell, that in blood preparations it is not uncommon to see only the granules; the cell itself may go to pieces, leaving an aggregation of granules.

In the normal blood there are no myelocytes, but these are frequently found in leukæmia. What are the characters of a myelocyte? It is not present in normal blood. At one time it was described as a very large cell, but it has been shown by many observers that these cells may be small or they may be large, so that we do not lay much stress on their size. But we do lay stress on the fact that the nucleus of the cell tends to be at one end, or, as it has been described by Cabot, that the periphery of the nucleus and the periphery of the cell are parallel to one another for a portion of the extent of the nucleus. To put it another way, the nucleus is situated eccentrically. The nucleus may be large and round; not uncommonly it is kidney-shaped or reniform, and the protoplasm

of the cell is accumulated mainly at one pole; and there it may, and usually does, contain eosinophile grains. In a myelocyte the nucleus is at one end and the granular protoplasm is at the other end of the cell.

The expression eosinophile cell, particularly with reference to leukæmia, is used unfortunately, because some people talk of these myelocytes as eosinophile cells, whilst others allude to the cell described above. That is why the classification, based on the character of the nucleus, is, in my opinion, of more value than one based exclusively on the character of the granules.

We now pass on to the subject of leukæmia. First of all as to what one understands by leukæmia. From the beginning you must remember that leukæmia is a particular kind of anæmia, a fact which is very often forgotten; and that the most serious symptoms in leukæmia are often not dependent on the leukæmic element at all, but on the anæmic element. I think one can very soon impress that by stating that the average proportion of red corpuscles in cases of leukæmia is about half the normal, the average being three millions. I think I am correct in saying that this anæmia is a characteristic feature which is usually present *throughout* the clinical course of the disease, whether the disease be treated or not treated. The second characteristic is that the white corpuscles are increased, and inasmuch as the white corpuscles are increased in a great number of other conditions, you have to qualify that statement in some way. Formerly the line was drawn at about 100,000 to the cubic millimetre, and if you had about ten to twenty times the normal number of white corpuscles present, the condition was talked of as leukæmia, and not as leucocytosis. There are two very much more important points, namely, that in cases of leukæmia the proportion existing between the different kinds of white cells is altered; and in the second place, in the most common form of leukæmia a large number of cells appear in the blood which normally are not present. Therefore leukæmia is something very different from a simple increase in the number of the white corpuscles. Further, one must draw attention to the fact that the increase in the white corpuscles is not necessarily present throughout the course of the malady. You may have cases of leukæmia with no excess of white corpuscles at a

particular period, and this occurs quite apart from the effects of treatment. But although that may be the case, the qualitative changes probably always persist; and that is why in the diagnosis of leukæmia it is so necessary to stain the blood; it is not enough to simply count the number of white corpuscles.

The next point I must touch on in describing leukæmia, still from a blood point of view, is that cases are divisible into two groups. I must repeat that it is simply a classification based on the blood examination, because there are various other classifications. Leukæmia is divided, according to the blood examination, into lymphocytic leukæmia and the so-called spleno-medullary leukæmia. All we mean by lymphocytic leukæmia is that the increase in the white corpuscles is mainly dependent on the presence of an abnormal number of lymphocytes; you do not necessarily mean that it is leukæmia which is dependent solely or mainly on disease of the lymphatic structures of the body. The increase in lymphocytes may be so enormous that whereas in normal blood these lymphocytes only constitute 30 per cent. of the white corpuscles, in lymphocytic leukæmia they may be over 90 per cent. There was a case in this hospital recently in which the proportion was 96 per cent.; that is to say, nearly all the white corpuscles in the blood were lymphocytes. Spleno-medullary leukæmia, on the other hand, is the form in which the increase in the white corpuscles is dependent mainly on an increase in the multipartite polynuclear corpuscles, and in which there are in addition large numbers of myelocytes. I pass round a drawing of an instance of that, and if you will compare it with this other one showing the lymphocytic form you will at once see that they are very different. In the spleno-medullary type the myelocytes may form as much as 30 per cent. of the increase in the white corpuscles; and that is how the notion has come about that leukæmia is characterised by the presence of myelocytes. To a certain extent it is true, but you must remember that you do not always get a notable increase of myelocytes, and this is more especially true of acute lymphatic leukæmia, and these myelocytes are seen to a small extent in the blood in other diseases.

What I have just given you is a division of the subject according to the blood examination; and

I may say at once to avoid repetition that if you have to deal with any form of leucocytosis, the increase in the corpuscles is an increase restricted to the normal polymorphonuclear cells of the blood,—that is to say, a totally different thing to what is seen in leukæmia. Even in the leukæmia in which the polynuclears are increased absolutely they are relatively diminished. Normally 60 or 70 per cent. of the white corpuscles are so-called polynuclears or polymorphonuclear cells, and 30 per cent. consist of lymphocytes; in the spleno-medullary form of leukæmia you may have 30 per cent. of lymphocytes and 10 to 15 per cent. or even 30 per cent. of myelocytes; it is obvious that this reduces the percentage of polynuclears to less than what is present in normal blood, although the actual number present may be vastly increased. That is why, in talking of leukæmia, you must carefully differentiate between the total number of white cells and the percentage. In leucocytosis, whether dependent on anæmia or on malignant disease, or other disease somewhere in the body, the great increase that may occur is practically entirely confined to these so-called polynuclear or polymorphonuclear cells; and, further, it is quite exceptional for the increase in white cells to be at all comparable to that seen in leukæmia.

There is one other remark I should like to make, namely, that although myelocytes are present in abundance in spleno-medullary leukæmia, you must remember myelocytes are present in the blood in other diseases, and more particularly in pernicious anæmia, and they are scanty or absent in some varieties of acute leukæmia.

Although myelocytes are present in the blood in pernicious anæmia, they are not so plentiful as in leukæmia; so, although you may find some myelocytes you do not find them in abundance and associated with a great increase of white corpuscles. If in a given case you do find myelocytes associated with increase of white corpuscles, you know you have to deal with a case of leukæmia.

There is another distinction between spleno-medullary leukæmia and lymphatic leukæmia, and that is in the spleno-medullary form nucleated red corpuscles are present in abundance, but in the other form they are not present to the same extent.

To recapitulate, there is not only a quantitative

but also a qualitative change in the blood in leukæmia, and the qualitative change is very much more important than the quantitative change; for the reason that in leukæmia it is not uncommon for the excess of white corpuscles to vary in amount and even to disappear. If the excess of white corpuscles disappears the qualitative change still remains, and you may have a case, such as one which was in the children's ward recently, in which the most careful counting of the blood failed to show any notable increase in the white corpuscles. But the blood contained plenty of myelocytes, and the patient had been in the hospital some time before, and recognised as a case of leukæmia, since there was then a notable excess of white cells. That shows you the value of the method of staining the blood.

We now pass on to the consideration of the disease from a clinical point of view. If you classify leukæmia from a clinical point of view, leaving the microscopic appearances of the blood quite out of consideration, it is divisible into acute leukæmia and chronic leukæmia. I do not propose to discuss the question of acute leukæmia to-day, because it is a disease which is very rare, and hitherto undescribed in this country. There are only about forty cases on record so far as I know. No cases have to my knowledge as yet been described in this country, but, as many of you know, a series of four cases has occurred in the practice of this hospital, and these will shortly be published. All I will say about acute leukæmia is that it is a disease which superficially simulates some varieties of purpura, and is one in which you do not get any notable enlargement of the spleen, although there is enlargement of the lymphatic glands and the bone marrow is affected; further, there is intense anæmia with an hæmorrhagic diathesis—bleeding at the mouth, rectum, &c. In the cases that occurred at this hospital, the disease was first detected accidentally owing to a blood examination having been made, and the clinical features of the cases are so striking that the recognition of the later cases was easy. There is nothing about a case of acute leukæmia that would suggest that it was one of leukæmia until you examine the blood. All the cases which have been recognised abroad have been cases of acute lymphocytic leukæmia—there has been no case on record, so far as I know, of acute spleno-

medullary leukæmia. The remainder of my remarks will apply to chronic leukæmia, as the cases of acute leukæmia referred to above will be described elsewhere shortly.

There is an old classification of chronic leukæmia which I must say I think is the best, though, perhaps, it cannot be said to be absolutely scientifically accurate. It recognises three varieties: (1) splenic leukæmia; (2) lymphatic leukæmia; and (3) medullary or myelogenic leukæmia. It is a classification which does not meet with favour, but it is really a very sound classification provided you understand that all that is meant by a splenic leukæmia is a leukæmia in which a marked post-mortem phenomenon is enlargement of the spleen, and that what you mean by lymphatic leukæmia is a leukæmia in which a marked post-mortem phenomenon is enlargement of the lymphatic structures, not necessarily glands; and finally, by myelogenic you mean that in which the post-mortem lesion is restricted to affection of the marrow of the bones. The reason the classification is scorned by some is because in every case of leukæmia, whether acute or chronic, splenic, or medullary, the marrow of the bones is diseased. That is quite true, but what you mean by medullary or myelogenic is that in which the marrow of the bones is the only structure diseased. That is the form of leukæmia which was described by Neumann some years ago, and cases of it have been recognised infrequently since. It is rare, and need not detain us any longer. Most cases of chronic leukæmia fall, then, into two divisions—those in which the enlargement of the spleen is the marked phenomenon, and those in which the enlargement of the lymphatic structures is the marked phenomenon. There is one other proviso which must be made. When one says the enlargement of the spleen is the marked phenomenon, one does not mean that it is the only phenomenon, as in all cases of leukæmia the abdominal and mesenteric glands are diseased. Of course the mesenteric glands are not accessible to clinical examination, hence the condition is often not recognised during life. But clinically there are those two types—one in which the patient comes to you on account of a large spleen, and the other in which he comes with great enlargement of the lymphatic glands. It is to this extent an accurate classification, but if you try to associate different blood conditions

with these two clinical varieties, and to say that in the splenic type you have the blood changes I have described above as characteristic of the spleno-medullary form, and that in the lymphatic type you have excess of lymphocytes, it is no longer true. The drawing I pass round is that of the blood of a patient who came into this hospital two years ago with a huge spleen; the enlargement of the lymphatic glands was quite moderate, and the case would have been lectured on clinically as one of spleno-medullary leukæmia; but the blood examination showed that the excess of white cells in the blood consisted mainly of lymphocytes. Therefore, in the present state of our knowledge, one cannot say that an increase in the lymphocytes in the blood is exclusively associated with enlargement of the lymphatic structures, and limited to these, and that an increase in the number of myelocytes is associated invariably with splenic and medullary disease.

I need not say that the class of cases in which the spleen is greatly enlarged without great lymphatic enlargement is the more common, and it is very much the more common, most people would say three out of every four cases, certainly quite two out of three cases.

Now a very few words as regards the morbid anatomy. In *all* cases of leukæmia the marrow of the bones, as far as I know, is diseased, particularly the marrow of the long bones, and the affection of the marrow takes one of two types. You may have the fatty marrow replaced by the "red currant jelly" marrow, or the fatty marrow may be replaced by gelatinous puriform material. As far as my experience goes the red currant jelly form is the more common. In all cases of leukæmia, as far as I know, the thymus is enlarged or persistent, and it is not uncommon for the thymus to be the seat of a definite lymphatic growth, a kind of mediastinal tumour. I believe that in all cases of leukæmia the mesenteric glands are diseased. The foregoing three facts apply to all cases—both the acute and chronic, the lymphatic and splenic forms. But after that you begin to get variations. In many forms the spleen is enormously enlarged, it may be ten pounds or even eighteen pounds in weight. Ten pounds is an ordinary weight for a spleen in advanced splenic leukæmia. In 30 per cent. of the cases the accessible lymphatic glands are enlarged, I mean the cervical, inguinal, axillary,

and commonly, I believe, they are also soft, in which respect they differ somewhat from those of Hodgkin's disease, in which malady they are frequently hard. The liver, of course, is frequently enlarged in cases of leukæmia, and perhaps this enlargement is more marked in cases of spleno-medullary leukæmia. This enlargement of the liver may be enormous, and the liver and spleen together may seem to fill the abdomen.

These are the principal gross post-mortem signs. The other fairly obvious post-mortem signs are first of all the peculiar colour of the blood, which is described as *café au lait*. I well remember seeing a case which had not been diagnosed during life, but which was diagnosed in the post-mortem room by the colour of the blood. Others have been struck by the bulky character of the blood-clots in the heart post mortem. In pernicious anæmia there is very little blood to be found; but that is not so in leukæmia, and it is a remarkable fact that the blood-clots are often singularly bulky. Petechial hæmorrhages are common in the serous membranes, particularly in the pleura and the pericardium, rarely in the peritoneum. I suppose the pericardium is the most commonly affected. Another post-mortem lesion, of which I have seen three instances, is hæmorrhages into the muscles. That is not a very common post-mortem sign. In one of the three cases I am referring to the hæmorrhages occurred into the rectus abdominis; and in another the patient really died from hæmorrhage into the psoas, which ultimately burst through the psoas and entered the retro-peritoneal tissue. Another interesting post-mortem phenomenon is the extraordinary deposit of uric acid which is sometimes seen encrusting the mucous membrane of the pelvis of the kidney and the ureters. Patients with leukæmia sometimes pass an enormous amount of uric acid, and I have seen uric acid coating the mucous membrane of the pelvis of the kidneys, and along the entire length of the ureters to the bladder.

I wish now to make a few remarks about the clinical course of the disease. All I shall say about acute leukæmia is that it is a disease which usually runs its entire course in less than eight weeks. Chronic leukæmia runs a very much more protracted course. Indeed, we cannot say how long it lasts, because nobody has ever seen a very early case. But usually it lasts for some years after it is first diagnosed.

The first thing I should like to draw your attention to is the extraordinary fact that patients with leukæmia will very often have quite a good colour; it is one of the most striking phenomena about the disease. You may have a patient with a tremendous amount of white corpuscles in the blood, who will nevertheless have quite a healthy, rosy face, and I particularly impress that fact, because even with people who are keen about blood diseases it does not always occur to them to examine the blood in patients who have a good colour. If you take any of the other blood diseases, such as Hodgkin's disease, chlorosis, purpura, or pernicious anæmia, the patients have marked pallor of the face, whereas leukæmic patients often have a good colour, perhaps not throughout the course of the illness, but at the time at which they first come under observation, and when the disease is fully established.

Now what do the subjects of leukæmia come under observation for? Probably in most cases they come either for pain in the splenic region, or for swelling in the abdomen, or on account of general weakness. The bulk of the patients come under observation for one or more of these three phenomena. The pain may be due to local peritonitis, owing to the formation of infarcts. The swelling of the abdomen is generally due to the enormous spleen. Among the cases of leukæmia which one has seen are many where the appearance would lead one to suspect ascites, owing to the size of the belly. Another symptom which such patients come for is dyspnœa. I have known leukæmia to be mistaken for asthma. The dyspnœa of leukæmia is very remarkable, by reason of its extraordinary paroxysmal character. If you have not seen such a case you will scarcely believe it. Patients with ordinary genuine asthma have very regular paroxysms at night, and may even have them at the same hour of the night for many nights, like the onset of pain in patients with gout. There was a patient with leukæmia in one of the wards here some two years ago who had serious dyspnœa every day in the late afternoon, and so bad were these attacks that each one was thought to be the patient's last. You can understand, therefore, that such cases might easily be mistaken for asthma. Ordinarily the dyspnœa of leukæmia is not so paroxysmal, and is brought on by exertion. Another group of symptoms

for which these patients may come is excessive and progressive loss of strength. I remember very well one case which I saw some years ago, in which I thought before examining the patient from the man's symptoms and his loss of strength that he had phthisis, but it turned out to be really a case of leukæmia. Lastly, patients may come under observation for renal colic, and I may again quote a case in this hospital where the ureters were encrusted with uric acid, and the patient came to the hospital on account of renal colic. He was found to have leukæmia.

I think I have said enough to show that the disease has no typical symptoms. It is very protean in its manifestations, and the diagnosis can only be made by a routine examination of the blood.

Now a few words as regards diagnosis. Provided you examine the blood you cannot very well make any gross error. If you do examine the blood, there are, as far as I know, only two errors which I think you may make. In the first place you may hit on a case in which there is no great excess in the white corpuscles at the time of examination; in that case you will not diagnose the disease unless you stain the blood. If the patient comes with a huge spleen, and you find no notable increase of white corpuscles, you may think it is a case of so-called splenic anæmia, a case of malaria, or a case of syphilis. But if you stain the blood, even when there is no excess of white corpuscles, you may yet find qualitative changes, which I have insisted upon, and you may find myelocytes. Personally I cannot help thinking that some of the cases of splenic anæmia, especially in adults, which have been described, may have been cases of leukæmia which happened to have been seen at a time when there was no increase in the number of white corpuscles. Although it is well known, it is not universally recognised that you may have leukæmia without there being at a particular time any excess of white corpuscles.

The other mistake which you may make, even if you do examine the blood, is that you may confound leucocytosis with leukæmia. There are some forms of leucocytosis—for instance in advanced malignant disease, or in cases of very large abscesses—where you may get a great increase in the number of white corpuscles, and the increase in the number of corpuscles may be comparable to the increase that occurs in some stages of leu-

kæmia, but you can distinguish it at once by staining. If you have to deal with leucocytosis you have an excess of the polymorphonuclear cells, whereas if you have leukæmia you have excess of lymphocytes or myelocytes. Unfortunately the staining of the blood is not often done, and there are very serious errors made in the diagnosis of leukæmia, some of which I will quote.

One of the most serious mistakes is to confound leukæmia with abscess of the liver. It is a mistake which may lead to the death of the patient, and it is one which has been made. Leukæmia is not uncommon in tropical climates, and patients with leukæmia have a big liver and a big spleen, so have patients with tropical abscess; in both also you get anæmia, and in both you get fever. In patients suffering from leukæmia a mere puncture of the liver with an aspirating needle may cause a hæmorrhage which will not cease.

I may quote another case which I know of, of a patient in another town in which the diagnosis of ovarian tumour was made. Laparotomy was performed, and it was found that the patient had leukæmia, not an ovarian tumour. This patient bled to death. These, then, are two very serious mistakes. Another serious possible mistake is to confound leukæmia with hydronephrosis. Bear particularly in mind that if you operate on a patient with leukæmia there may be hæmorrhage, like that seen in hæmophilia, which cannot be arrested.

As regards mistakes which are very serious from the medical point of view, if not from the patient's point of view, a greatly enlarged spleen may be confounded with a pleuritic effusion. Such an error is made every now and then, and I have seen it occur. Such a mistake can be obviated by an examination of the heart, and also, of course, by careful examination of the abdomen and chest, quite apart from the examination of the blood. Leukæmia may also be confounded with Hodgkin's disease, but that is not a matter of very great importance. Still, in the true Hodgkin's disease there is diminution in the number of red corpuscles, and only a very slight leucocytosis, nothing comparable to what is seen in leukæmia. I do not mean to say that those I have mentioned are the only mistakes which can be made in diagnosis, but they are the most serious of them.

Now a few words as to treatment. Of course you know that operative treatment is out of the

question. Although certain forms of enlarged spleen can be successfully removed, in leukæmia such a course is contra-indicated, mainly on account of the hæmorrhage. The only drug that is known to have any marked effect on the disease is arsenic. Arsenic, however, has not anything like such a marked effect in leukæmia as in pernicious anæmia. A great deal of the improvement which is put down to arsenic in leukæmia may occur in the ordinary course of the disease. I do not wish to make light of the value of arsenic in leukæmia, but you cannot always be sure that the improvement observed is due entirely to that drug.

An important point which I had forgotten to mention in the clinical history is that variations in the size of the spleen may and often do occur. You must remember that the spleen may vary enormously from time to time, and not only is that the case, but the spleen may return to practically its normal size. Diminution in the size of the spleen, so far as I know, does not necessarily mean a good prognosis; the spleen may diminish in size coincidently with an improvement in the patient's condition, but it may also diminish in size as death approaches. The case I referred to with periodic paroxysmal dyspnœa was a case in point. Although he had an enormous spleen when he came in, when he died the spleen was almost normal in size. Under the administration of arsenic the spleen not uncommonly diminishes in size. Then, again, the beneficial influence of arsenic is very often shown more by the diminution in the number of white corpuscles than it is by an increase in the number of the red corpuscles. That is very remarkable. In pernicious anæmia the administration of arsenic is followed by a great increase in the number of red corpuscles, whereas in leukæmia arsenic may cause a disappearance of the increase in the white corpuscles without the return of the red corpuscles to their normal number. Quinine has also been used in the treatment of this disease, and oxygen inhalations have been tried. I only mention oxygen to say that in my opinion it is not of any use at all in diseases in which the difficulty of breathing is due to the loss of hæmoglobin, and where the dyspnœa does not arise from any difficulty in the air reaching the blood. The best treatment of leukæmia is undoubtedly the administration of arsenic in increasing doses.

MEETING OF THE SOCIETY OF ANÆSTHETISTS.

At 20, Hanover Square, March 17th, 1898,
The President, Dr. DUDLEY BUXTON,
in the Chair.

(Continued from p. 92.)

MR. STEPHEN A. COXON read the following paper, entitled—

"The Continuous Administration of Nitrous Oxide."

MR. PRESIDENT AND GENTLEMEN,—In dental practice the want of a more prolonged anæsthesia is often felt when there are several teeth to be removed. In 1888 I commenced to try if anæsthesia could not be lengthened by allowing a small stream of gas to pass into the mouth by means of a bent tube. This proceeding certainly lengthened the anæsthesia. Encouraged by this, I employed a larger tube—such a one as I now show,—with the result that there were few dental operations that could not be completed with one inhalation of the gas, and in many there is no sign of returning consciousness as long as the injector is kept going. The method of administering is to anæsthetise the patient in the ordinary way with nitrous oxide; then on removing the face-piece to place the curved tube into the mouth until it is a short distance from the uvula (for preference the injector is passed to behind the prop); then, with the gas-bag well filled up, a steady stream of gas is maintained. The patient with every inspiration inhales a mixture of gas and air, and is of necessity kept under for a longer period than would otherwise be the case if he were only inspiring air. There is one thing upon which stress must be laid, viz. that the gas-bag be kept well filled with gas, so that the gas comes out under pressure, or otherwise only partial success will be obtained. It is of necessity rather wasteful, but the results you get will more than compensate you for this.

One day, when giving gas in this fashion, I found the patient's mouth unusually cold. This undoubtedly arose from the stream of nitrous oxide gas that was being sent into it. On the patient leaving I tried by placing the bulb of a thermometer in the gas-bag. The result was that, in a

room registering 64°, the thermometer in the bag was a shade under 50°. Since that time I have made a practice of letting the gas traverse a small copper coil as it passes to the bag, and of placing the coil in hot water. This method heats the gas and prevents it causing possible trouble to the lungs of a delicate patient.

Statistics.—With reference to statistics, I have the record of the past ten years. The following cases may be mentioned:—A female of about 22. Period of anæsthesia, 80 sec.; operation completed. Female aged 24, of hysterical temperament; period of anæsthesia, 60 sec.; operation completed. Female, aged 28, 1 min. 10 sec.; operation completed. An anæmic boy, 1 min. 10 sec.; operation completed. It is needless to remind you that during the operation it is very necessary to keep the mouth as clear as possible of blood.

Effects upon the patient.—They as a rule get up and go away, as they would do under an ordinary gas extraction. During the time of using the injector there is very little jactitation, and the patient is nearly as quiet as he would be under chloroform. Now, after using the tube for a while you can make it of great service to the operator; it will act as a tongue depressor, and obstruct the entrance of teeth into the throat that have slipped out of the forceps; also it will push the tongue out of the way of the forceps. During no part of the time, after once getting the patient under, do you obstruct any of the air-passages. As the anæsthesia lessens you can renew it by simply opening or closing the stopcock of the gas-bag.

The PRESIDENT said they were very much indebted to those gentlemen who had brought the subject before them,—Mr. Coleman's system, which supplied gas through a mask fitted over the nose, and Mr. Coxon's system with an instrument in the mouth. Through the courtesy of Mr. Coleman he had had an opportunity of trying his apparatus in about twelve cases, and he was greatly struck with the success which followed its use, even in his inexperienced hands, for he had never touched the apparatus before. He was able to secure anæsthesia for four to four and a half minutes, while fourteen teeth were being extracted, and he certainly could have gone on longer if the patient had had the good taste to require more teeth taken out. He had adopted a

slightly different method from that advocated by Mr. Coleman, as he only applied the nose-piece after removing the face-piece. He thought it would be better for them in the discussion to confine their attention to the nitrous oxide apparatus, without going into the larger question of the employment of chloroform or ether. He used to use a catheter passed through the nose extensively in the administration of chloroform, but he found that a large percentage of people had so much blockage of the nostril that a good deal of time was usually lost in introducing the catheter; and he came to the conclusion that it was a distinct drawback to employ a catheter through the nose. Then, he thought, they ought to place on record the very important point that, even though they might have a prolonged anæsthesia, it was their duty to warn their dental friends, which he did with due submission in the presence of Mr. Coleman, against multiple extraction. Occasionally multiple extractions were important and necessary, but he was sure that as a rule the loss of blood which was sometimes so strikingly demonstrated in multiple extracts left results which were most detrimental to the well-being of the patients.

Mr. HERBERT PATERSON said that more than two years ago he made some experiments with a view to prolonging anæsthesia by the mouth, and for that purpose he employed a catheter through the nose. Although he found that as far as the anæsthesia was concerned he could get a considerable prolongation, the method, for other reasons, was so unsatisfactory that he gave it up, and had not tried it since. He found hospital patients were very liable to bleed at the nose through the catheter being passed. Another practically unsurmountable barrier to the method was the almost impossibility of properly sterilising the catheter. They could not use a metal one, and could not boil a flexible one. Some weeks ago Mr. Coleman, hearing he was interested in the subject, very kindly lent him the apparatus which he had shown them, and he had been able to use it in some seventeen or eighteen cases. All he could say was that from his short experience with it he could not commend it too highly in dental work. He had maintained anæsthesia in two cases for exactly five minutes. A most important case he would call to their notice—that of a nurse. It was absolutely necessary for a

long period of anæsthesia to be maintained. The nurse had broken off a tooth some months before, leaving one of the fangs, which had given rise to an abscess, and the root of the tooth was buried in the abscess sac. She was given gas and oxygen, and although she was given a good dose, the anæsthesia was not sufficiently long to enable the dentist to even localise the root. After a short interval, therefore, he gave her gas by Mr. Coleman's apparatus, and she was kept under for 4 min. 10 sec., and a successful operation performed. This was a case in which no other anæsthetic was sufficient unless chloroform or ether were used. The nurse afterwards, in answer to questions, said she had not noticed any difference in going under the anæsthetic in the two cases, but she came round quickly after the administration by Mr. Coleman's apparatus. With Mr. Coleman's permission he would venture to indicate one or two possible improvements. Mr. Coleman himself would admit that the details of the apparatus were only experimental, and he (Mr. Paterson) thought that some modifications might be made with advantage. In the first place the nose-piece was of rigid material; he (Mr. Paterson) was having one made of celluloid on the principle of Dr. Silk's celluloid face-piece, and by this means, he thought, pressure marks on the patient's face could be avoided. He had found the bag rather in the way, and would suggest that there should be only a small reservoir bag at the back of the patient's head, and that the main bag should be on the floor, out of the way of the operator. The face-piece also might be made of celluloid, and should be rather larger, and made to fit the nose-piece more accurately, which would thus be more readily kept in place. In all the cases in which he had used the apparatus the anæsthesia was stopped simply because the operation was at an end, and he could have gone on considerably longer. Mr. Coleman had spoken of the importance of maintaining a pressure of gas, which he thought was a point requiring special attention. It was true that in maintaining pressure a considerable waste of gas ensued; this, however, was inevitable if satisfactory anæsthesia was to be obtained.

Mr. COLEMAN, in reply, expressed his thanks to the gentlemen who had spoken. The apparatus he had shown was nearly all home-made, and

therefore, as Mr. Paterson had said, was capable of considerable improvement. At the present time, not holding any medical appointments, he could not make any experiments with it himself, and he had to express his great thanks to the President, to Dr. Hewitt, and to his friend Mr. Paterson for having kindly tried these experiments. Regarding the employment of a tube in the mouth, that had been adopted for many years by the late Mr. Clover, but the tube used by that gentleman probably had not the same internal diameter as Mr. Coxon's tube, and therefore the results were almost *nil*. He (Mr. Coleman) had taken care that the nose-piece should be applied in the first instance, and he thought there was an advantage if an elastic band encircling the head was used such as he showed. He thought it was right to restrict the discussion, as suggested by the President, but he would like at some future time to bring the apparatus he mentioned for giving chloroform and air in definite proportions before their Society. He would remark how much superior was Mr. Clover's method over the others in the fact that a deep inspiration could be taken without any tendency to irritation of the glottis.

Mr. COXON, in reply to the remarks on his paper, said: As to the injector being in the way of the operator, when the user got accustomed to it it did not seem to be in the way. In extracting lower molars, for instance, it was very necessary for the dentist to change over as well as the anæsthetist. He had managed to keep himself out of the operator's way, and to manipulate the tube as well. Probably the tube might seem somewhat of a nuisance until the user got expert with it. Regarding the cold gas, the objection he had to it, and therefore the reason he introduced the apparatus for warming it, was that the mouth got cold during the administration, and it was not desirable for cold gas to be pumped into the lungs. He agreed with the remark that multiple extractions should not be done if they could possibly be avoided.

The PRESIDENT proposed a vote of thanks to the gentlemen who had contributed papers that evening. They had to thank two gentlemen who were not members of the Society, one of whom had come a considerable distance, and the other had come under circumstances which made his attendance a special act of courtesy to the Society. Mr.

Coleman had been honourably associated with dentistry for very many years, and was perhaps the *doyen* of the profession.

NOTES, ETC.

On the Effect and the Therapeutic Uses of Alcohol in Childhood.—C. Baron ('Der aertzl. Practiker, 1897, Nos. 14 and 15) relates first the harm occurring to children from the administration of alcoholic beverages in health, passing then to its medicinal value when exhibited in disease, and asserts that only very few indications for its administration can be found, the most important indications being the affections of the respiratory apparatus, bronchitis, pneumonia, œdema of the lungs, diphtheria—which diseases are accompanied by high fever or by a great demand on the functions of the heart and lungs, when the various properties of the alcohol combine in producing its well-known favorable effects; for here we not only make use of its stimulating qualities on the cardiac function, its influence in increasing the number and depth of the respiratory act, and the lowering of blood-pressure, but also of its power to reduce the temperature, and to economise albumen. In erysipelas, surgical fever, great loss of blood, &c., alcohol is frequently indicated. The number of cases in which the undesirable effects of alcohol predominate are however much more numerous. Above all do diseases of the brain and of the nervous system fall into this category, beginning with simple forms of nervousness, and passing through the more severe forms of epilepsy, chorea, &c. In like manner is alcohol contra-indicated in heart disease, nephritis, cystitis, enuresis, in rheumatic and similar affections, in diseases of the liver, of the gastro-intestinal tract, and in all chronic conditions.

Baron does not support the view of Grosz, who looks upon chronic disturbance of the nutrition as an indication for the use of alcohol. In these cases also we had better avoid the use of alcohol, especially as these anæmic, scrofulous, rachitic children are frequently extraordinarily sensitive to even small doses of alcohol. Baron has observed, after the administration of a saccharine solu-

tion of iron and manganese, in a number of children, cephalalgia, insomnia, &c., due to the cognac contained in it for its preservation, while other preparations of iron which did not contain any alcohol could be administered with impunity. We are well able to do without alcoholic beverages in chronic diseases, and are well supplied with plenty of valuable reconstructives in the various milk preparations, whey, eggs, malt preparations, iron, quinine; while stimulants, beef-tea, bouillon, coffee; in gastric affections, Russian tea, in addition to those in our pharmacopœia are plentifully to be found as substitutes.

In those cases, however, in which alcohol is indicated, we may usually rely on the heavy wines (Tokay, port wine, madeira, sherry, marsala); cognac and rum will only be necessary in patients who have been previously addicted to beer and wine. The dose should be regulated, according to Ewald—twenty drops for a three months old child; fifty drops for one six months old; one teaspoonful for nine months old; one and a half teaspoonfuls for a twelve months old; one children's spoonful for patients two to three years old, three to four times daily. Children suffering with diphtheria are able to bear much higher doses.—*Pediatrics*, May, 1898.

The Tongue as a Clinical Guide in Disease.

—A recent number of the 'Indian Medical Recorder' gives the following in regard to the tongue:

A broad, pallid tongue, with a loaded base, says atony, and refers you to a want of action of the entire viscera below.

The remedial agents would be cathartics and tonics, especially those mild but effectual in character.

A shrunken tongue, pinched in expression, indicates functional inactivity of digestion, and requires great care in choice of food as well as quantity. In this condition of tongue we have atony also.

It is the tongue of advanced fevers, inflammations of the mucous membranes, and want of assimilation, hence great caution both of remedies and food. Here we must not use cathartics. Mild aperients may be carefully used.

A contracted, pointed tongue, with dryness and dark fur, is the usual tongue of typhoid fever and

other low grades of fever, when all thinking minds would use great care in the treatment and food.

The dryness or moisture of the tongue denotes the extent of the disease of the intestines, and will point us in that direction.

A fissured tongue points to the kidneys, either an inflammation or something wrong with secretion.

Yellow coatings are usually associated with morbid liver and want of biliary secretions, and would indicate mild hepatics and tonics.

Raised papillæ, bright red, denote irritation of ganglionic nerves and irritation of stomach, especially the mucous coating. Shows exhaustion; no digestion, and needs rest; nux vomica twenty drops, and the food to be warm and taken in small quantities. Bismuth and pepsin after food.

Broad, thick tongue, papillæ not visible, but looking raw, denotes a septic condition of blood, and favours typhoid fever. Indicates, if deep red, sulphuric acid; if pale, sulphite of soda. Liquid food, sipped warm, in small quantities.

Deep, dark red tongue and dark coating indicate septic condition of blood.

Shades of dark brown and black denote typhoid condition or septic conditions.

Pale, dirty fur on tongue denotes acidity, and a septic condition of system; indicates sulphite of soda; but if membranes are deep red, sulphuric acid will be admissible, because it would show an alkaline condition of blood.

Contracted, pointed, inability to hold still, and drawn to one side of mouth, denotes trouble with the nerves, and perhaps the brain. Requires great care and study of condition.

Dry tongue always denotes feverishness or inflammatory condition, affection of the nerve-centres of ganglia.

Thick tongue, and curved edges upward, denotes atony of the nerve-centres of ganglia, requiring stimulants, nux vomica or strychnine and quinine.

Pointed, narrow tongue is the tongue of sluggish condition of digestion and assimilation and congestion, especially of the base of brain. Restlessness and constant change of position are usually present.—*Medical Age*, May 10th, 1898.

Cure of Pruritis Vulvæ by Operation.—

Von Mars ('Monats. f. Geburtsh. u. Gynäk.'), in three cases of pruritus vulvæ under his observation,

noted that the labia majora were, probably from changes due to swelling or atrophy, in a condition of entropion, hair being turned inward on the vestibule and clitoris. When the hair was carefully trimmed the pruritus at once ceased. Von Mars suggests the formation of an artificial ectropion of the labia in these cases, by the removal of an elliptical piece of skin from the outer limits of the labium majus.—*Cincinnati Lancet Clinic*, May 7th, 1898.

Atrophy of the Uterus following Internal Application of Steam.—Baruch ('Centralblatt für Gynäkologie,' No. 5, 1898) reports a case which illustrates that the intra-uterine application of steam is not altogether free from unhappy results. The patient, a young woman of 27, was treated for continuous hæmorrhage. Menstruation returned four months after giving birth to a child, and continued for eight weeks.

An intra-uterine application of steam was made, and the hæmorrhage immediately arrested. But no subsequent menstrual periods occurred, and the woman passed through the usual symptoms attendant upon the menopause.

Ovarian extracts were then given for some time, but produced only abdominal pains, without re-appearance of menstruation.

Two years later an examination was made, and the uterus found to be small and hard. The external os was so obliterated by cicatricial tissue that a probe could not be passed, and the ovaries could only be indistinctly felt.—*University Medical Magazine*, May, 1898.

The Spastic Night Cough with Vomiting and Coryza in Young Children.—According to Dr. P. Gastow ('Der Kinder-Arzt,' 1897) this very common affection is of reflex origin, sometimes due to intestinal parasites, sometimes to other causes, but usually the result of a coryza posterior. The cough has the following characteristics.—it is spasmodic, similar to whooping-cough; it causes nausea and vomiting, and it occurs at night when the child is in bed, never when it is up, and rarely by day. It occurs only in very young children, because they do not expectorate and cannot clear the nose; the mucus therefore drops backward into the pharynx, and the irritation of the pharyngo-laryngeal

mucous membrane thus produced causes a reflex cough. The cough is spasmodic, because the mucus collects in the region of the arytenoids and the vocal cords, causing glottic spasm and attacks of suffocation. The cough occurs at night, because then the position of the head favours the gravitation of mucus to the pharynx and larynx. Treatment consists in the application three or four times daily, through the whole length of the nasal cavities, of tampons saturated with borovaseline. This at first causes sneezing, but later the infant bears it well. The snuffing causes the vaseline to flow into the pharynx. Astringents, such as tannin or alum, and especially antipyrin, may be added to the vaseline.—*Medical Record*, May 14th, 1898.

Saccharin as an Intestinal Antiseptic.—After a review of the work hitherto done in search of intestinal antiseptics, Dr. Descheemaeker ('Echo médical du Nord,' April 10th) records a series of experiments upon rabbits, and subsequently upon the human subject, both in health and in disease, to ascertain if a rapid diminution of intestinal ferments could be obtained by the daily employment of saccharin. The saccharinate used was Monnet's No. 3, and is a saccharin of sodium, containing, however, ninety per cent. of pure saccharin. It was given in doses of from fifteen to thirty grains once daily about two hours before the principal meal. The author concludes that his experiments recorded show that the saccharin used by him must take rank among the best intestinal antiseptics. The results, both on rabbits and on man, are constant. In all the experiments the ordinary germs of the intestine, and especially the *Bacterium coli-commune*, were markedly decreased in numbers. The ingestion of the remedy was well borne by the sick, and the daily analysis of their urine never displayed a trace of albumin or any other abnormal product, while the urea remained constant throughout the experiments.—*New York Medical Journal*, May 14th, 1898.

A Tumour in the Femur resembling the structure of the thyroid gland was recently removed by Goebel from a woman of fifty-four years, who had had femoral hernia twice and pseudo-arthritis. This makes the sixteenth case of struma metastasis on record.—*Munch. med. Woch.*, April 12th.

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A CLINICAL DEMONSTRATION ON PULMONARY CAVITIES IN TUBERCULOSIS,

BY

DR. WETHERED,

At the Brompton Hospital, February 16th, 1898.

LADIES AND GENTLEMEN,—The following remarks concerning pulmonary cavities in connection with tuberculosis refer partly to their formation and partly to the prognosis and diagnosis of the different conditions under which they occur. For the purpose of orderly consideration in a demonstration of this kind the older divisions of phthisis, as it used to be called, into first, second, and third stages still forms a very convenient *clinical* classification. It cannot, however, be called a true *scientific* classification, because it is not possible to have a lung in the third stage without the first and the second stages being also present. We can only say that there are three distinct periods of pulmonary tuberculosis—there is the deposition of tubercle, with consequent consolidation of the lung; there is the breaking down, or disintegration; and there is the existence of a cavity. Destruction of lung tissue is the essential anatomical feature of pulmonary tuberculosis. All cases, unless happily arrest takes place, tend to the formation of a cavity by destruction of lung tissue.

There are four varieties of cavity to consider, viz. the recent cavity, the quiescent cavity, and (for purposes of further division) there is what is known as the secreting cavity and the ulcerous cavity. By far the great majority come under the heading of recent cavities or of quiescent cavities.

The *recent cavity* is the first result of the breaking down of caseous nodules in the lungs. Whatever the derivation or constitution of a pulmonary solidification may be, if the process is not checked liquefaction ensues, and this process will probably take place in more than one centre. These centres may be scattered through one or both lungs, or

more commonly the caseous masses are close to one another in the apex of one lung. The cavity thus formed is at first very small, then the neighbouring cavities coalesce, the walls between being broken down. In the breaking-down process bridges of lung tissue are left between the cavities, and you may have a complete network of lung tissue leading from cavity to cavity. Sometimes the excavation is very extensive, as will be seen by some of the specimens I have placed on the table.

We do not have any physical signs from a cavity until a communication is effected with a bronchus, resulting in the expulsion of the softened contents. It is difficult to state what size a cavity must attain to in order to give physical signs for diagnosis. The usual statement is that we cannot detect with any certainty a vomica smaller than a walnut, and this cavity must communicate freely with a bronchus.

Sometimes we are puzzled when a patient comes to us and on examination no signs whatever of a cavity are found, and yet a few days after we do find signs of a cavity of no inconsiderable dimensions in the same patient. Sir Richard Douglas-Powell draws attention to this sudden formation of a cavity, and gives the following explanation ('Diseases of the Lungs and Pleura,' 4th edit., p. 416):

"It not unfrequently happens that we fail to get distinct evidence of pulmonary softening for some time after troublesome but more or less dry cough and hectic symptoms point very strongly to its presence. There may be dulness, harsh breathing, and some fine spongy crepitation increased after cough, but none of those distinct clicks characteristic of pulmonary softening. The patient will suddenly, in the course of the night perhaps, expectorate a considerable quantity of purulent matter, and we find evidence—cavernous râles, &c.—of the existence of a cavity. The explanation of these phenomena is obvious enough. A nodule of consolidation of appreciable dimensions becomes uniformly caseous, and then softens in its centre and gradually liquefies throughout before communicating with a bronchus, when its fluid contents are at once expelled, and auscultatory evidence of the existence of a cavity becomes abruptly developed. In the post-mortem room we may often cut through such softening nodules in all stages of ripeness for exit. They sometimes undermine and rupture through the pleura, and may well be designated caseous abscesses."

The explanation of these cases is that every element for a cavity is there except the communication with a bronchus.

Professor Rindfleisch very lucidly explains the manner in which these softened masses finally communicate with a bronchus. There is a general traction upon the bronchi during inspiration, and as soon as the softening process is sufficiently advanced a separation is effected between the root of the softened mass and the bronchus which passes into it. Air is immediately drawn into the rent, passes behind the liquid contents, which are expelled through the bronchial tube, and the air takes its place.

If we diagnose the formation of a cavity we can obtain corroborative evidence by the examination of the sputum; not only can we say by this means that a cavity exists, but we can say if it has existed a long time and whether the process is acute or chronic. We can state from examining the sputum without seeing the patient that that patient is suffering from pulmonary tuberculosis (from the presence of tubercle bacilli), also that the patient has had the disease for at least eighteen months or two years, but at present he has an acute outbreak and destruction is going on. That diagnosis is made by the elastic tissue. The old method of boiling with caustic potash is troublesome, and requires a long time to carry it out, and also tends to destroy those characteristics I am going to comment on.

It is easy to obtain everything one wants in the way of elastic tissue without the process of boiling. Throw the sputum into a flat dish, and with a pair of forceps and scissors pick out the little yellow opaque masses which are so constantly present in the sputum. It is important to distinguish these opaque particles from food. I have seen masses picked out that are simply globules of milk. The nodules that we want are yellowish, and not so white as food matters. You may find three distinct varieties in arrangement of the elastic tissue in the sputum, and I have frequently seen these three several times during one demonstration. I have also seen two of the different kinds under the same field of the microscope. The most common form consists of small pieces with clear-cut edges branching dichotomously. If these are present we may conclude that destruction is going on and the condition is subacute. In other cases you may find a large mass branching dichotomously and

forming a rough cast of the alveoli. Then we know the process is going on very acutely.

The third kind consists of small pieces of elastic tissue, but instead of having a clear-cut outline the edges are blurred. If a little acetic acid is run under the cover-glass fine bubbles will be formed, and the outlines of the elastic tissue will become apparent. This irregular formation is due to a deposit of lime salts on the elastic tissue, and it must have lain in the cavity for from one to two years, showing a very chronic process. In very acute cases it is common to find the two last kinds together, and on asking the patient you will find a history of two or three years' illness, with intervals of improvement, and then an acute outbreak of the disease. The examination of sputum, therefore, helps us in prognosis and in diagnosis.

The temperature chart also helps us very much in prognosis. The temperature chart in cases of recently-formed cavities is always hectic in type, and it is important what time of day the temperature is taken. When taken at eight in the morning and eight in the evening we are apt to lose the height of the temperature, which is nearly always to be observed at five in the afternoon; therefore I generally request that the temperature should be taken in the morning and at five in the afternoon in the cases of pulmonary tuberculosis, and of course, if necessary, a third time in the evening.

We now come to the quiescent cavity. A cavity may at once cicatrise, and after a time only a scar is found, or it may become quiescent. It may gradually contract from cicatrisation, or it may become quiescent and contract, or it may continue secreting indefinitely. The statistics as regards the cicatrisation of cavities have only been brought out during the last two or three years.

Dr. Fowler and Dr. Martin from statistics obtained from the records of the Middlesex Hospital found that in a large number of cases dying of diseases other than phthisis, upon examining the lungs in a certain proportion obsolete tubercle was present. That these were instances of obsolete tubercles was proved to be so by Dr. Martin inoculating guinea-pigs with the material.

From 1879 to 1886 Dr. Fowler found that out of the total of 1943 deaths 177 had obsolete tubercle in the lungs; showing a percentage of 9.

In looking back at the histories of these cases

we very rarely find that the patient gave any history of an attack of phthisis. That is a point in prognosis, and I have found it often encouraging with patients to tell them that so many people have tubercle in the lung which has spontaneously obsolesced.

Dr. Martin took two years, 1890 and 1891, and found out of a total of 445 deaths that 42 had obsolete tubercle, yielding 9.4 per cent.

For the purpose of some figures I am getting out I have looked through the post-mortem records of 1887, 1888, and 1889 at the Middlesex Hospital. In these three years there was a total of 785 autopsies; in these there was obsolete tubercle in 124 cases, giving 15 per cent. of cases of obsolete tubercle found in the lung; in a large number of cases, more particularly male cases dying from all forms of cancer, obsolete tubercle was found in the lung.

In a good number of these cases there were found patches of obsolete tubercle scattered through both lungs in all lobes. These masses were very considerable, and in some cases which I have abstracted, evidence of obsolete tubercle was found in the apices of both lungs, so that in even the very worst cases where tubercle is scattered through both lungs we need not expect necessarily a fatal event.

When a cavity becomes quiescent the walls become condensed and toughened by the development of fibrous tissue so as to shut it off completely from the surrounding lung. The contents become less and less abundant, the sounds become drier until no moist sounds are heard. Then this kind of cavity gradually shrinks in size, the chest wall flattens, and the opposite lung encroaches towards the affected side, and to make up for loss of space, emphysema forms round the cavity.

Now I pass on to the secreting cavity, and this is a condition which is extremely difficult to treat. It is one of the most trying kind of case for medical men to deal with. The secreting cavity is one of tolerably old date which has ceased to extend, and is unaccompanied by active pulmonary disease. The patient may remain wasted, with no temperature. The cavity is thick-walled, and is lined by a smooth, opaque, pyogenic membrane, which can be easily scraped off, exposing a highly vascular dusky red subjacent surface. The trabeculae, which are numerous, present a similar appearance.

Such cavities may go on indefinitely secreting a diffuent creamy pus, yielding gurgling sounds, with marked amphoric breathing and a dull percussion note. There is no fever, or it is only very trifling, consisting of a slight rise of temperature at night. The tongue is clean, with a tendency to redness and a loss of epithelium. The appetite is not as a rule very poor, but the patient slowly loses ground and acquires the sharp, hungry, pinched face peculiar to chronic phthisis, with clubbing of fingers and toes. There is also a tendency to albuminoid degeneration of the organs. Diarrhoea may supervene, and troublesome sickness, especially with cough and laryngeal complications, occur. Prognosis is bad, and these cases are very stubborn to treatment. Occasionally the secretion dries up and the cavity becomes quiescent; as a matter of fact, however, that patient is sitting on the brink of a volcano. A cautious prognosis should be given. One accident likely to happen to these people is a fatal hæmoptysis; the patient may be getting on well, hæmoptysis occurs, and the patient dies in a few minutes.

In regard to ulcerous cavities, these are formed in the usual way, and they may be for a certain time quiescent, or merely secreting for some time, when, owing to some unfavorable influence, they assume a state of active ulcerative extension. They are angry looking, deep dusky red on the inner surface, often studded with hæmorrhagic points or ulcerative nodules. They are highly tuberculated and irregular in shape, but are sharply differentiated from the lung tissue by a thin vascular wall. They contain a copious blood-stained purulent secretion, which when expectorated is mixed with the ropy secretion from the intensely vascular bronchi which communicate with them. The lung tissue surrounding the cavity is injected and oedematous, and at distant points of the lung may be found pneumonic centres, which evidently owe their origin to the inhalation of the acrid secretions from the cavity (Douglas-Powell). In such cases there is high fever present, with rapid, quick pulse and a furred tongue, and a tendency to typhoid symptoms. The sputum is generally mixed with blood, or dark clots can be removed from the cavity; sometimes hæmorrhage occurs from the erosion of a large blood-vessel.

It is difficult to say what are the physical signs necessary to make a diagnosis of a cavity. First of

all, on inspection there is loss of movement on the affected side, and there is usually a certain amount of falling in of the chest wall locally. This latter sign generally frightens the patient, but you may, however, assure him that it is a favourable thing; it means, as a rule, contraction owing to formation of fibrous tissue slowly surrounding the cavity. The percussion note is usually dull, but it may be tympanitic if a large cavity exist, and we sometimes can get a dull note or a tympanitic note, according as the patient shuts or opens his mouth. With the mouth open the note becomes more tympanitic; with a cavity which is very large indeed there may be a high-pitched tympanitic note. Owing to the development of emphysema we may be deceived, and sometimes on percussion we can hardly tell any difference from the normal side.

On auscultation the breath-sounds are always altered; we may have bronchial, cavernous, amphoric, or tubular breathing. I make use of all these terms because unfortunately our nomenclature of physical signs has become decidedly mixed; what some people call tubular breathing others call bronchial or cavernous, while others have introduced another term—broncho-vesicular. I think it better to confine the word tubular breathing to what we hear over croupous pneumonia.

By bronchial breathing I mean breath-sounds in which inspiration is harsh; there is a distinct pause between inspiration and expiration, and expiration has the same pitch as inspiration. The varieties of breathing are chiefly characterised by the difference in the expiration; as a rule in normal breathing it can be scarcely noted, whereas in bronchial breathing the inspiration is as loud as expiration.

In cavernous breathing there is a pause between expiration and inspiration, and expiration is of a different pitch to inspiration. Some authors say it is a higher note, others a lower note. I think they are both right. I think that much depends on the musical ear of the observer. Amphoric breathing is generally described rightly as the sound of blowing over a bottle.

As a rule, in a recent cavity there are always adventitious sounds present, and it is one of the cardinal rules for examination of the chest never to examine a case without making the patient cough. I remember hearing a case demonstrated

to a class as a case of what the lecturer called a "typical dry cavity;" but the patient coughed just before the students listened, and that demonstration failed. The râles are of a very characteristic nature, they are medium in size, and distinctly bubbling, and if disintegration of lung tissue is going on very rapidly there are other peculiar high-pitched sounds known as "humid clicks." Then there is another sign which we hear in very large cavities which is called "metallic tinkling;" it is exactly like water dripping into a well, and has a peculiar echoing sound. It is one of the diagnostic points between excavation and consolidation of the lung; in the latter the râles are finer.

There is another sign which I have not seen described in books; it is what is called post-tussive suction; on the patient coughing you have a peculiar sort of suction sound immediately after the cough. It is very characteristic of the formation of a cavity. On clicking two coins together over the chest Dr. Habershon believes that one can map out the limits of a cavity. Pectoriloquy is also present.

If you have all these sounds then you may absolutely diagnose a cavity.

Sometimes the lung is consolidated; there may be what is called caseous pneumonia, in which the whole lung becomes rapidly consolidated from the deposition of caseous material. You have tubular breathing rather than bronchial breathing, and fine crepitation instead of the medium-sized bubbling râles.

There is another form which is known as "pulmonary tuberculation." It is a very rare form of tubercle in which the whole of one lobe becomes consolidated; but on listening over it you hear tubular breathing without any moist sounds; this changes into bronchial breathing, evidencing the formation of a cavity. This variety consists of a deposition of tubercle in the lung, but it crumbles away something like lupus on the cutaneous surface.

The difficulties in diagnosis of a cavity consist chiefly in the development of emphysema round a tubercular nodule and a simple want of care in not asking the patient to cough.

As regards the prognosis of pulmonary tuberculosis, it depends on the variety of the cavity; if recent, we cannot say what is going to happen. We judge a good deal from the temperature chart;

if we find the temperature not above 101° at night, and normal in the morning, if pressed we may give a fair diagnosis. When, however, the temperature does not fall to normal in the morning,—for instance, 103° at night and 101° in the morning,—then the prognosis is grave. The worst form of temperature chart to meet with is when the temperature is high in the morning and low in the evening.

Another very important point is to ascertain the state of the opposite lung: supposing there is a cavity in one lung and the other is affected, then the prognosis is not good; if the lung is, however, expanding well over to the diseased side, then that is a good diagnostic point.

The Extract of Supra-renals as a Stimulant in Dangerous Chloroform Narcosis.—

Dr. F. A. Magnkovsky, in the 'Russian Archives of Pathological Anatomy' ('American Medico-surgical Bulletin,' May 10th), presents the following conclusions which he has reached after a series of observations made upon dogs for the purpose of testing the action of the supra-renal extract upon these animals when they had been narcotised by chloroform almost to the point of arrest of the heart and respiration.

1. The intra-venous injection of the supra-renal extract is capable of saving the life of dogs suffering from extreme chloroform narcosis.
2. Compared with the procedures of other investigators, notably those of Schüller, Laborde, and of König Maas, intra-venous injections of the extract are preferable on account of its more rapid action.
3. Extract of supra-renals exercises a marked influence upon the respiration, the blood-pressure, and the tone of the heart muscles even in such small amounts as from fifteen to thirty grains of a 1 per cent. solution. Hence it should be borne in mind that it is a powerful remedy, and should not be given in large doses.
4. During chloroform narcosis it is wise to have prepared a fresh solution of supra-renal extract, preferably sterilised by boiling, in order to controvert any sudden collapse.
5. The best results, in cases of imminent death due to chloroform, are obtained by means of combined procedures—such as intra-venous injections of supra-renal extract, massage of the cardiac region, and the subcutaneous injection of physiological salt solution.

New York Med. Journ., May 21st, 1898.

THE SURGICAL TREATMENT OF ACUTE SEPTIC PERITONITIS, LOCALISED AND DIFFUSED.

Address delivered before the South-West London Clinical Society, Wandsworth Town Hall, March 9th, 1898,

BY

C. B. LOCKWOOD, F.R.C.S.Eng.

MR. PRESIDENT AND GENTLEMEN,—Although I have spoken and written about this subject on various occasions* before, yet I am quite sure you will pardon me for bringing it before you again, not only because it is of great importance, but because I venture to think it is an exceedingly difficult subject, and one which at the present time is in considerable need of enlightenment. Not that I am going to provide that enlightenment, but I propose to discuss this evening certain cases of peritonitis which have occurred, and all of which, I may say in passing, were due to infection from the interior. I hardly think it necessary to refer to cases in which the peritonitis is obviously due to infection from the exterior,—for instance, by gunshot wounds and wounds made by people who are attempting suicide, and so forth; nor do I propose to refer to peritonitis which is met with accidentally whilst performing operations on people with mechanical obstructions, such as bands, and in cases of intussusception.

Before I proceed let me say that for the present one has to use the word peritonitis without always having a very clear notion of what the word peritonitis means in a given case. It may be said at once that you may have two kinds of peritonitis at least. For instance, I will pass round a drawing made from a histological section of a case of peritonitis, in which you will see that the peritoneum, and more particularly the subperitoneal fat and connective tissue, is full of strings of micrococci. The surface of the peritoneum is covered with lymph and has micrococci upon it too. Here is another section of an acute peritonitis where there is a large mass of bacteria, but only on the surface. The infection in the first case spreads in as far as the subperitoneal fat, whilst in

the other the infection is all upon the surface of the peritoneum, and has hardly penetrated into the substance at all.* I have no doubt there are a great many other kinds of peritonitis, but offhand I give you these two very separate and distinct kinds, and as I proceed you will see that these differences have a very great influence upon the treatment and prognosis of the particular case. Whatever is done to a peritoneum in which the whole of its substance is infected and in which the subperitoneal fat is full of streptococci, it is obvious that no application or washing out of the peritoneal cavity is likely to have a very marked effect upon the course of the disease. Furthermore, it is much more likely that in the first case, in which the whole peritoneum is infected, that the whole of the person is likely to be infected with bacteria; in other words, such patients are more likely to die of acute sepsis or acute septicæmia. In the second case, where the infection is all upon the surface of the peritoneum, and has hardly penetrated its depth, it seems reasonable to suppose that measures applied to the peritoneum could possibly produce some result. Now it is to some extent possible clinically to draw distinctions between the two kinds of infection of the peritoneum, and if those distinctions can be drawn between the different kinds of infection, obviously some grounds for prognosis and treatment can be obtained.

Now the cases that I am referring to to-night are cases of peritonitis of the most acute kind. I have said that there are cases of localised and cases of diffused septic peritonitis, but it must be obvious to anybody that every case of diffused septic peritonitis must have a local beginning and must for a time be a localised septic peritonitis, and it is at that stage of localisation that it is possible by means of surgery to obtain results. Before I end I shall show that cases of very severe diffused septic peritonitis have been cured by operation. I shall show also that the earlier the operation the greater is the possibility of a successful issue.

Taking a case of very acute septic peritonitis, I would say that from a surgeon's point of view the classical descriptions are hardly of very much service. I may be quite wrong, but I cannot help thinking that if a surgeon is going to wait until the

* 'Med.-Chir. Trans.,' vol. lxxviii, 1895; also 'Clinical Journal,' April, 1896.

* These sections are figured in the speaker's 'Lectures on Traumatic Infection,' Figs. 3, 4, and 5, p. 14, *et seq.*

classical signs of peritonitis become pronounced or obvious, the time for his services is probably passed; and that brings me to a point which I wish particularly to emphasize, namely, that to cure cases of peritonitis by operation it is absolutely essential that they should be diagnosed at the very earliest possible moment. The difficulties of diagnosis are exceedingly great, but my own mental condition is that with greater experience of cases, and more particularly operative experience, the more improved become one's powers of diagnosis.

First of all, as regards the general condition of a patient with peritonitis. As far as I can see, one of the traps into which most people fall is this: that to a most extraordinary degree they are misled by the general condition of the patients. As to the appearance of the patients, it is said that persons with peritonitis have a certain facial aspect—*facies Hippocratica*. So they have when they are moribund; but that is not the stage at which the surgeon cares or desires to intervene. The next point is that such patients are very deceptive in their voices. I have been very much struck by that. I remember a man dying of peritonitis, with his abdomen full of pus, with hands and feet cold, sit up in bed, and with a perfectly strong voice reply to questions and call out for people to get things for him; and when asked if he had any pain he moved quite rapidly in bed from one side to another and turned himself over for his loins to be examined. You can understand that if you were to be guided by the aspect and voice and general appearance of such patients you would be very much deceived. But patients in such a condition as that I have described, or even prior to that, present signs which should certainly enable you to say that they are doomed. Now one of the symptoms which I have learnt gradually to attach more importance to than any other is the condition of the pulse. I do not know why it is, but, with very rare exceptions, I do not think a human being can have an acute inflammation of a small part of the peritoneum, let alone a large area, without the pulse being markedly accelerated. I was much impressed by this in the case of a medical man on whom I successfully operated eighteen months ago. I saw him at three o'clock one afternoon, and he had a pulse of 97. He had passed flatus, and I think he had had the bowels opened. His vomiting was less

than previously, so that there appeared reason for some delay; but I made the remark that he should be watched, and if his pulse accelerated and went over 110 the operation should be performed. As a matter of fact, within four hours his pulse was 120. The abdomen was opened and an abscess evacuated; a faecal concretion was got out, and it was found that he had a perforated appendix. I have been told of a case in which a man with septic peritonitis had a slow pulse, but he had some peculiar heart disease, which accounted for the small increase in the pulse-rate. I have a recollection of seeing a woman with septic peritonitis who had a slow pulse, but they are peculiarities.

Next as to temperature in these conditions. I think there is a great deal to be learnt from the temperature in peritonitis. You are aware that it is commonly said that the temperature is subnormal in peritonitis. But I think more often there is a rise of temperature; indeed I cannot help thinking that if you are careful to look for it in the right way you always get a rise in temperature. In going over my notes I found a case of peritonitis in which the patient had an abdomen full of pus and was moribund. That might have been passed off as a case of acute septic peritonitis with subnormal temperature, but as a matter of fact, although his temperature in the mouth and axilla was subnormal, in the rectum it was 103°. I had a still more striking case exemplifying the point that the common ideas as to temperature in peritonitis may not be particularly correct. A woman who had been shot in the abdomen was under my care. She had several perforations of the duodenum, and I think of other parts of the intestines also. She afterwards got a very acute form of peritonitis, and I remember that her temperature in the mouth and in the axilla was 99°, whilst her rectal temperature at the same time was 105°. Therefore if any one were to tell me that he had seen a case of peritonitis with a subnormal temperature, I should be obliged to ask him where the temperature was taken. I believe that if the temperature were always taken in the rectum it would be found that in all cases of peritonitis the temperature would be raised.

Now, coming to the abdomen, if I were asked what was one of the most constant symptoms of acute septic peritonitis, I should say acute intes-

tinal obstruction. Nearly all the cases I have seen—the exceptions are very few—had at some period or other acute intestinal obstruction. It is said that cases of peritonitis have no obstruction at all. That is true. I had a case under my care a little while ago which was never sick and had no signs of intestinal obstruction, yet her pelvis was full of pus. But before the end of the disease she had very acute intestinal obstruction, which was the probable cause of her death. All people with peritonitis, at some period or other, cease to empty flatus and fæces out of their bowels. The explanation which I have been accustomed to think for myself, is that the functions of the intestines are first of all to deal with their contents in the way of digestion and absorption, and next to propel them. The intestine is very much like a knee-joint or hip-joint, which, when it becomes inflamed, ceases to move—so the vermicular movements of the intestines cease, and the contents are not propelled, when its serous covering is inflamed. When I first began to operate upon the subjects of peritonitis, I operated upon them more by chance than by design, because I operated upon many of them thinking they were cases of mechanical obstruction. Then I went through another stage of thought, and wondered whether they were cases of mechanical obstruction or of acute peritonitis. Now one is able to say in most cases after careful investigation that this is a case of acute septic peritonitis with marked symptoms of obstruction. Continuing with the abdomen, rigidity is a point upon which much has been said, but I cannot help thinking that by the time the abdomen has become very rigid the case is almost outside the reach of surgery. Much rigidity of the abdomen seems to me to mean very marked and extensive peritonitis. In many cases where the abdomen has been very rigid I have observed that inflammation has extended far beyond the peritoneum. I have very clearly in my recollection a case which I have recently seen, in which the abdomen was markedly rigid; it was also red and œdematous, and the case was, as it proved, beyond the reach of operation. Before the rigidity of the abdomen occurs there are other signs which I think would generally lead one to know that the person had got peritonitis without waiting for such a forbidding sign as rigidity. First of all, the abdominal distension is very

significant. The intestines cease to propel their contents, the bacteria in the intestines still continuing to make gas, the abdomen soon begins to distend. The intestinal bacteria manufacture gas when they are growing in test-tubes and in gelatine. Therefore in most cases, when the abdomen is opened and infection has come from the interior, some gas escapes. This production of gas in the peritoneal cavity has also a very practical bearing, because I have more than once opened an acute peritoneal abscess which had misled people because the abdomen was resonant. Now it is hardly necessary, after what I have said, to say why many of these peritoneal abscesses contain not only pus but a large amount of gas, and afford a resonance which may mislead those who are not acquainted with its cause. Again, I have been very much struck with the immobility of certain parts of the abdomen in these cases. If a person has an inflamed appendix, the point of the abdomen over that spot is immobile. That immobility very often points offhand to the seat of the septic peritonitis. Another point of great importance—which Mr. Greig Smith has called attention to very well—is the extreme stillness and silence of the abdomen. You know that if the abdominal wall is at all thin you can see the coils of intestine; in peritonitis they never move. If the affection were mechanical obstruction or colic (I have known colics due to copaiba and sandal-wood oil mistaken for obstruction), the intestines are on the move. As soon as the intestines become inflamed they are still. Furthermore, when you listen with the stethoscope in a case of peritonitis nothing whatever is heard. If in a doubtful case the observer were to fail to listen for this stillness he might fall into error easily. Of course, with the stethoscope in mechanical obstruction or intestinal colic all sorts of noises are heard.

There is one other point about the abdomen in peritonitis which is well worthy of attention, and that is the pain which the surgeon or the attendant can himself elicit. Those pains are of extreme importance, because they are the guide to the operation. For instance, in the last two cases I have seen the patients referred their pain to the umbilicus, as they nearly always do, but pressure upon the right linea semilunaris elicited very acute pain; and in both of these cases the appendix was

perforated with abscesses around it. But supposing pain is not always to be felt over the linea semilunaris, there is another place in which it should always be felt for, and that is in the rectum. I am always afraid when I see a case of peritonitis, to ask, except very guardedly, what has been felt in the rectum, because the precaution is so often omitted; but in a large number of cases I am sure a rectal examination will clear up the diagnosis. When the finger is passed in the rectum and pushed up beyond the bladder the patient utters a shrill cry, so exceedingly painful is it. The surgeon who does that may feel an indurated or elastic swelling. If it be a female, vaginal examination will give useful information. The uterus may be fixed, or there may be extreme tenderness in Douglas's pouch. In a great many of my female cases of acute septic peritonitis the sepsis has been traceable to the Fallopian tubes; and it is easy to understand that you might elicit no pain or tenderness in the front part of the abdomen, but very considerable tenderness and pain would be evident upon vaginal or rectal examination. Again, in boys it very frequently happens that the appendix lies in such a way that its root is in the iliac fossa while its free end is hanging over into the pelvis, so that the pus collects in there. I have seen very many cases which were considered doubtful because the rectal examination had been omitted. They say it should be peritonitis because of the distension and pain, and rapid pulse, but they cannot find any tenderness or any lump. They have not found the tenderness or the lump because they have not made a rectal examination. An attack of retention of urine or acute pain on micturition is often associated with this pelvic type of appendicitis, and this sign ought always to be sought for. Further, I have seen a pelvic abscess in this acute septic peritonitis sometimes mistaken for a full bladder. This may seem strange; but it is not so strange to those who have seen the cases, because sometimes a pelvic abscess feels almost exactly like a full bladder. The only way to settle the matter is to draw off the water and see if the swelling still persists. I think I have observed one curious thing about these pelvic abscesses in acute peritonitis, and that is they often tend to bulge not only in Douglas's pouch but underneath the left linea semilunaris.

You will observe that till now I have referred

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mainly to cases in which there has not been much mass or swelling in the iliac fossa. In those cases of peritonitis there is not the same difficulty about diagnosis. In the kind of case to which I have chiefly been referring the abdomen has been distended and tense, but there has not been a lump, and a tender spot has only been elicited with difficulty, or by examination *per rectum* or *per vaginam*.

As regards the intestinal obstruction, I have always observed that there have been great difficulties about that question. The passage of a motion is, of course, a clear matter; if a patient has passed a motion he has not got intestinal obstruction. But there are degrees of intestinal obstruction—what I refer to is the absolute non-passage of flatus or fæces. People with peritonitis sometimes pass a little flatus, but that ought not to mislead you. When there has been a passage of a little flatus, in order to clear up the diagnosis I have been in the habit of ordering an ordinary enema of soap and water with castor oil and turpentine. If that is efficiently administered, and fails to cause the bowels to act or bring away flatus, I do not hesitate to act upon the information conveyed by such failure, provided other symptoms coincide. But in these cases it is of supreme importance that some person should be able to say definitely whether the patient has passed flatus or fæces, or both, because it makes all the difference in the world. If one is told that the patient has passed flatus, and he has not, it may cause delay, which the patient may pay for with his life.

Now there is no doubt in my mind that an early operation in most cases of acute septic peritonitis would yield comparatively good results. Remembering that the cases about which I am talking are inevitably fatal, and weighing against that the reasons for performing laparotomy upon them, I would be inclined to think of it in this way:—If a surgeon who practises modern methods were about to open the abdomen of a healthy adult (these patients are not advanced in life as a rule), the risk of opening the abdomen would be a very minor one, the risk of death being, I should say, about 1 in 500 or less. Indeed, I think the chief risk which the patient would incur would be a ventral hernia if care were not sufficiently exercised. The risk of leaving these cases of which I am speaking

alone, on the other hand, is so great that they always prove fatal, and generally by the fifth day. I know there are cases of chronic localised septic peritonitis in which the patients will go for a long time and perhaps recover, but they do not come within my range this evening; I am dealing with those showing an exceedingly rapid pulse, a distended abdomen, constant vomiting, and intestinal obstruction. Supposing that the diagnosis of acute septic peritonitis had been arrived at, either local or diffuse, it is of great importance that some kind of attempt should be made to ascertain the source of infection of the peritoneum. In most of my cases the source of infection has been the appendix, and in most of these there has been some indication for that—previous attacks or pain over the seat of the appendix at the time of examination, or something which has indicated some disturbance of the bowels; such, for instance, as a quantity of some curious kind of food. In one of my cases the patient had recently eaten a quantity of fish, and in another case a quantity of celery late at night. A common source of infection in these cases is the Fallopian tubes, and very often there is something which will help to establish that diagnosis. But in most cases, as far as I have seen, the nearest you can get to the source of the infection is to say it is situated below or above the umbilicus. Oddly enough, I have operated upon four people with perforating ulcer of the duodenum; but I think it is sufficiently practical to know that you have to deal with a case above or below the umbilicus.

Supposing you have made up your mind that it is a right and proper thing to operate on a patient with acute septic peritonitis, either localised or diffuse, remember that the operation, though important, is really only a part of the treatment. A great deal has to be done before the operation is undertaken at all. First of all, it is important to recognise the way in which most of these patients with acute septic peritonitis die. If I were asked how they die, I should say nearly all of them succumb to some heart failure; the pulse runs up from 100 to 120 or 140 for a few hours, and then suddenly ceases. During the operation, as far as I can see, one of the great troubles with the anæsthetist is to keep the heart going. On that account my usual practice is to give the patient a dose of strychnine before the operation; and it is a wonder what

large doses of strychnine patients with acute septic peritonitis will bear, one twentieth of a grain being a not unusual quantity before the operation. In addition to this they may have one fiftieth grain during the progress of the operation, and one fiftieth at its termination. Another point in the preparation of these patients is to fill the rectum full of hot brandy and water. That is a proceeding which cannot do any harm, and it helps them to get over the operation. In addition to these, to avoid shock it is very important to have the room hot, and the patient should, if possible, be put upon a hot-water bed, or surrounded by hot-water bottles during the operation. Another matter I should mention is the washing out of the stomach, which is sometimes desirable, because if the patient vomits up all sorts of materials during his anæsthesia he will be very uncomfortable, and may incur other dangers by getting the contents of his stomach into his air passages. In reference to the operation, I should say that one of the first things is to have all the appliances very hot. It is difficult to get the nurse to supply lotions, &c., sufficiently hot. She ought to be told to have her sponges and lotions at a temperature of 110°. That may seem rather warm to put inside a human abdomen, but I do not think it does any harm, because by the time the sponges are actually applied they have dropped to 105° or so. I believe this matter of heat is a very important one for these desperate cases.

Now as regards the performance of the operation, the person who undertakes it ought to do so with quite clear ideas in his own mind as to what he is going to do, because while the patient will survive an operation lasting twenty, thirty, or even thirty-five minutes, very few will survive one of an hour. The routine procedure in a case of very acute localised septic peritonitis is to cut down and open the abscess at once, not to waste any time in looking for the appendix or the tubes; but if there are not many adhesions the opportunity ought to be seized of removing a gangrenous appendix and certainly removing the concretion, but not to go out of your way to look for an appendix amongst a great many adhesions. Then we come to a point about the treatment of these diseases about which I think there may be some differences of opinion. Some surgeons confine their attention to cleaning out the abscess cavity

with swabs or sponges. In regard to that very much depends upon the case. If it is quite clear that there is a single abscess with well-marked walls I think no one would disturb them and open up fresh tracks in the peritoneum for the invasion of the pus. But in most of the acute fulminating cases there is not a distinct abscess wall. The other day I opened an abscess under the linea semilunaris, washed it out, and in moving the intestines aside I came to another and that was washed out; further on I met with another collection, and washed that out. So that it is quite clear that the surgeon should have thought out before he begins exactly what he is going to do in the way of looking for abscesses. I irrigate these abscesses out with extreme freedom, and treat an abscess inside the peritoneal cavity in the same way as I should an acute abscess in a person's arm. I think if anybody here was dealing with a very acute abscess in a person's deltoid, he would lay it open freely and wash it out thoroughly, and leave in a drainage-tube. For washing out the abdomen I have used comparatively strong solutions of antiseptics. For instance, at the beginning of this year I operated upon a policeman who was an exceedingly fat man, and had evidently been in the habit of consuming large quantities of beer. His abdomen was exceedingly tense, he was constantly vomiting, and he had very acute intestinal obstruction. The thing which chiefly guided me to the recognition of the acute septic peritonitis was the obstruction and the pulse, which was 120, and he had a very acute pain over his appendix. In addition to that, his temperature was 102°, so that it was quite clear that he was suffering from inflammatory disease. I made an incision over his appendix, and came upon an abscess and a perforated appendix. Turning over the coils of intestine I came across pus, and as I did not know how much pus there was amongst the coils, I washed out the peritoneum and put in a drainage-tube for his pelvis, and another in his iliac fossa. The result of that was very striking. I did another thing which is not quite orthodox, but which I believe is worth thinking over and ought to be considered—I ordered him to have 5 grains of calomel the moment he came out of his anæsthetic, and gave instructions that he was to have 5 grains more if that did not act, and he also had an enema. He had the two doses—10 grains in

all—and as the result his bowels were copiously opened. My reason was that he was very plethoric, and as the intestines were also very distended I thought it was his only chance of safety. The result of all this was that his temperature went down to normal, and remained there. His pulse did not improve, and when I saw him again after that, his pulse being still at 120, I felt anxious. His pulse began to improve afterwards, but I did not feel at all sanguine about him until his pulse slowed down. The acceleration of his pulse showed he still had some septic absorption, and that his heart was suffering from that. Therefore, I am now in the habit of washing out abdomens exceedingly freely with strong antiseptic, and I choose the biniodide of mercury because it is one of the strongest, and it does not combine with anything in the abdomen, and probably any that is put in comes out again. These patients do not get mercurial poisoning. Another point upon which the surgeon has to make up his mind is what to do in a neglected case, and in which there is a great deal of intestinal distension. In my earlier cases—which have been my worst—I have freely let gas out of the intestines with a fine trocar; I have also made cuts into the intestines to let out the fæces; and, strange as it may seem, these cases have got better, whilst others have not. If you cut the intestinal wall while you have it in sight, it is a perfectly innocuous proceeding. A surgeon has not achieved his task if he puts back intestines into the abdomen which are distended with flatus. I have not time to make reference to drainage in these cases, but I think india-rubber tubes are the most comfortable. If a glass tube is put in I think it should be replaced at the end of forty-eight hours by an india-rubber tube, which is gradually lessened.

Now, in many of these cases the struggle only begins after the operation is over. And the struggle usually turns on three things. First of all keeping the patient's heart going. It is well worth while flogging the heart along for a few days, and it may be done with strychnine and with brandy; it may be necessary to give ether, and so forth. The other trouble is with vomiting. Now these patients will vomit for a few days, and then it will gradually cease and they will get better. Any attempt to feed by the mouth aggravates the vomiting, and this does more and

more harm to the heart, and the patients succumb. Here the value of a good nurse also comes in. If a nurse is thoroughly competent at rectal feeding it is quite marvellous how a patient will go on with nothing by the mouth for several days until his chance of vomiting is over. Rectal feeding is quite an art, and I would say that it is worth anybody's while to study it, and I say that without pretending that I have mastered it. I say that rectal feeding in these extreme cases is all-important. The way in which I try to get this feeding done is first of all to get the rectum washed out with tepid water; and as these patients are usually very thirsty (it is one of their great troubles) it is a very good thing to instruct the nurse to leave half a pint of tepid water in the rectum after it has been washed out. This is absorbed, and goes towards relief of the patient's thirst. After twenty minutes an enema is put in. Whatever it is made of this enema ought to have certain characteristics. First of all it should be concentrated. Good milk and meat essence peptonised is very useful. Enemata should be carefully peptonised; too often the nurses are told that foods thoroughly peptonised are unpalatable, forgetting that for these purposes it has to be put where there is no taste. With these enemata a little brandy may be used, but the brandy must be good; bad brandy is exceedingly irritating to the rectum, and an irritated rectum will soon cease to hold enemata. To cause the enema to be retained it is a good plan to add five drops of laudanum. An enema of that kind, four ounces at a time, can be administered by a skilful nurse over a long period of time. Supposing the heart is managed properly and the vomiting is kept in abeyance, there is one other problem the solution of which often makes or mars a case, that is abdominal distension. This is most difficult to get over. I do not know whether I am right, but I have lately got into the habit of giving patients calomel, and following that up by enemata. I believe a great deal of what you will do depends on what you find at the operation. I have got thus far from watching cases as to think that you can give calomel much more freely in these cases without harm than you would suppose. I was taught that after strangulated hernia you must give patients opium to keep the intestines quiet, and that in peritonitis you must do nothing which will make the intestines stir. I have come to the con-

clusion that he would be a very clever man who could make the intestines stir in a case of peritonitis, and I cannot think that in a case like this, when it becomes a question of whether you will try to get the patient's abdomen flat or will let him die without doing anything, that a dose of calomel makes much difference. If you are afraid of drugs by the mouth a great deal can be done by enemata.

By far the best enema for getting flatus out of patients is the ordinary soap and water enema, with half an ounce of castor oil and half an ounce of turpentine. Remember that if these patients get sick their pulses become very bad. To avoid sickness or faintness after the enema, a dose of strychnine may be given. If those troubles I have alluded to are got over, you will find the pulse begin to fall, and the patient will probably convalesce. But every now and then cases will go on quite well for several days, and then will have a recrudescence. I had one which went on very well indeed for ten days, and then had a recrudescence and died. In such cases it has been a question in my mind whether a second operation should be performed. I think it depends very much on the kind of relapse that occurs. Patients require a good deal of watching to know when a relapse is coming on, but there is one thing which I think inevitably tells you; if the abdominal distension is never quite gone, and the temperature has never quite fallen to normal, one thing will tell you that something is wrong, and that is the pulse. I have been watching a lady who had septic peritonitis due to perforation of the appendix. Her pulse and temperature fell till the tenth day, and then she had a rigor, and the pulse went up to 120. Then she began to be a little sick, and then one observed that the lower part of the abdomen was a little tender. I gave her an anæsthetic, and found she had a collection of fluid in Douglas's pouch, and I opened a stinking abscess there. The pulse then began to slow down, and she convalesced. There are cases in which a second operation, not necessarily severe, may save the patient's life. Some of the last cases included in the statistics which I am about to give were of the most desperate kind—moribund, in fact. In some the abdomen has been distended, the pulse has been uncountable at the wrist, and the hands and feet cold, and yet I have operated

upon them and let out pus, removed the appendix perhaps, and washed out the abdomen, and I can imagine anybody of common sense saying what earthly use was the procedure. But I do not think a surgeon ought to think of himself altogether when he is dealing with a case of that kind. All I can say is I have seen such extraordinarily desperate cases recover that it is hard to think anybody has gone so far that they have not got a chance. Some time ago, in the 'Medico-Chirurgical Transactions,' I published a case of a boy who was in St. Bartholomew's Hospital, who had a high temperature, pulse 120 at the time of operation, and a very much distended abdomen. When the abdomen was opened it was simply full of pus. There were no adhesions, the intestines were covered with lymph, and it was altogether a case which ought to have been inevitably fatal. The cause of his peritonitis had to be found. His appendix was normal, the rest of the large intestine was normal. I then searched his ileum very rapidly and found a hole in it, which was the source of his peritonitis. The intestines were tapped with a trocar and the air let out, openings were made to let out fæces and sewn up again, his abdomen was washed out, and he recovered. That is such a striking fact that since then I have been afraid to say that no one has a chance of getting better from septic peritonitis. I may mention another perfectly desperate case, which astonished me very much. A lady was confined, and she had what somebody called puerperal fever. She had sepsis of the uterus and Fallopian tubes. She was treated with antistreptococcic serum, and within a month she got better and was allowed to get up. In the afternoon she was seized with violent pain in the abdomen, and immediately began to vomit and have diarrhoea. I saw her that night, and thought that I could still feel her pulse at the wrist, but the hands and feet were cold. She was continuously vomiting, had a distended abdomen and diarrhoea, and she said she was going to die. Obviously she was, but knowing what I did I did not think it was right to let her die with her abdomen full of pus. So we put her on a little truckle bed. I sat on a small stool on one side of the bed and Mr. Wadd, who was my assistant, knelt on the other side, and my friend Dr. Malcolm, a very skillful administrator, gave the anæsthetic. I came

upon an abscess in the left Fallopian tube, and found a little opening in it through which pus was spurting. Her peritoneum was injected and inflamed, and contained a large quantity of pus. By the time I had got that far Dr. Malcolm said he thought she was dead. But I thought it was a pity to let her die like that, so I got a tumbler of brandy and a hypodermic syringe, and injected her with brandy until I thought that she improved a little, and that she moved. However, Dr. Malcolm said, "she is still too bad to go on." We then got a big basin of hot biniodide lotion—1 in 4000—and poured it into her abdomen. This revived her, and I was able to pull the abscess up to the wound, open it, and fix in a drain. The wound was rapidly sutured, and then we cut her clothing off, covered her with blankets and surrounded her with hot bottles, and began to inject strychnine and brandy again. I did not see much of the after part of her case. She had a dreadful bout of vomiting, but she got quite well, and I met her in the street the other day. I then made up my mind that it is a little rash to say it is not possible to do anything for even a very desperate case of peritonitis.

As I was going through the matter I looked up to see how many of those kind of cases had got better. I went back to 1894, because I have definite notes from that time. Since then I have had 27 desperately bad cases of peritonitis, that is in people who were dying. One of the cases perhaps ought not to be included, because, although dying, it might by some be called subacute. However, I prefer to include it as the end was fatal. I opened a young lady's abdomen and found a stinking abscess which had gone on for some time. I thought she convalesced. I saw her at the end of three weeks, but I heard afterwards that she got a recrudescence and died. The cause of the peritonitis was never discovered; the appendix appeared to be intact, but that does not go for a great deal. But on the other side of the account I have put a similar case which got well, to balance it. I found that on one side I have 14 cases which died, and on the other side 13 which have got well, so that apparently each person in desperate straits has a 50 per cent. chance of recovery. I might say that amongst the deaths was one which died on the operating table, and I need not have operated upon him, but I did it because

I did not think it was right to let him die with his abdomen full of pus. In another case there was a perforating ulcer of the duodenum in a person who was moribund.

I may say in conclusion, that if we learn to approach this subject with unprejudiced minds, and without preconceived ideas, very much progress might be made in the early diagnosis of cases, and also in their surgical treatment.

Intubation in Laryngeal Obstruction. A. J. Wood.—The following points regarding intubation are emphasized:—The tubes must be correctly made. Previous practice upon a cadaver, a larynx, or animals is essential. In introducing the tube the left forefinger, protected with strapping, is passed back to the pharynx, then drawn forward between the arytenoids, lifting the epiglottis against the tongue. The tube is passed into the mouth with the handle of the introducer touching the chest, the handle then raised till the tube touches the left finger, when the latter is slipped behind the tube, the handle raised to a right angle, and the tube passed into the larynx; the finger should be placed on the top of the tube as the obturator is withdrawn. To prevent a young child from pulling out the tube by means of the silk, the elbows may be stiffened with cardboard held in place by strapping. The child must not be let to sit up. Nourishment may be administered with the head lying back over a pillow, semi-solids being often more easily swallowed than liquids. The nurse should be instructed to remove the tube in case of extreme dyspnoea, or if the silk has been bitten through to invert the child and express the tube by pressure from outside. Extraction, when it is necessary to use the extractor, is the most difficult part of the operation. The instrument should have a control screw, set so that the blades will open only widely enough to hold the tube that has been used. The author believes that intubation should be done with the patient in the lying position. He now uses chloroform anæsthesia for intubating and extracting. The retention of the silk in the tube is most necessary in order that the tube may be quickly removed should it become blocked with membrane. The steam tent is useful only when dyspnoea comes on after the removal of the tube. Regarding the length of time the tube should be worn, it is better to remove it early—after two or three days if antitoxin is used—and reintubate if necessary. We must remember that the return of the dyspnoea may be very sudden. — *Amer. Gynecological and Obstetrical Journal*, May, 1898.

MEETING OF THE SOCIETY OF ANÆSTHETISTS,

At 20, Hanover Square, April 21st, 1898,

The President, Dr. DUDLEY BUXTON,
in the Chair.

MR. GRANT MORRIS read the following notes of a case of death under an anæsthetic.

MR. PRESIDENT, LADIES AND GENTLEMEN,—Having had the misfortune of losing a patient after the administration of ether, I am placing before you the following notes of the case, firstly in order to elicit the opinions of members of the Society as to the cause or causes of the accident, and secondly because I think it presents some features of unusual interest. M. G.—, female, 30, married, admitted to St. Thomas's Hospital January 11th, died February 15th. The following notes of her condition up to the time of operation are abstracted from the clinical notes taken in the ward. Family history unimportant. Previous history: patient had had three children, one being still-born and the other two dying in infancy. During the last eight years she had had several attacks of severe pain in the left groin, running down the leg to the inner ankle, not followed by hæmaturia, nor accompanied by either vomiting or diarrhoea, the pain did not last more than half an hour, was always confined to the left side, and was not present in the region of the left kidney. Present illness: for three months prior to admission she had suffered from pain in the abdomen accompanied by vomiting. She had lost weight during this period. Before her admission she was attended by a medical man, who on testing her urine found it contained a large trace of albumin. On admission, a pale, cachectic woman complaining of vomiting and pain in the abdomen. On examination, heart and lungs normal, tongue furred and flabby, pulse 72, regular and strong; urine specific gravity 1024, acid, distinct trace of albumin, no sugar, blood, or pus. On January 14th, on examination *per vaginam*, above the vaginal vault on the left side, a hard, irregular, slightly movable body, in shape and size like a date stone, was felt, its position being thought to be

compatible with the supposition that it might be situated in the ureter.

After a week in hospital the albumin had disappeared from the urine. Her symptoms persisting, after consultation an exploratory abdominal section was performed on February 15th. Ether was the anæsthetic selected, and was administered from a Clover's inhaler. During the period of induction the index never exceeded two, and throughout the operation stood at one or a little under. She went under quietly in about three and a half minutes; there was no struggling or excitement, no coughing or laryngeal spasm. Anæsthesia was normal in all respects, pupils equal and moderately contracted, no conjunctival reflex, respiration free and unimpeded, pulse full, soft, and regular. Towards the end of the operation,—that is to say, in the last quarter of an hour or so,—there was some cyanosis and a good deal of secretion of frothy mucus, but neither the amount of cyanosis or secretion was sufficient to cause alarm, or more than I have witnessed on many occasions before, both in my own patients and those of others, without any evil effect ensuing. The operation, which had occupied sixty-five minutes, having been completed and the dressings applied, the patient was transferred from the operating table to the trolley ready for her removal to the ward; at this time she was still somewhat cyanosed; though rather shallow and sighing, the breathing was impeded as though vomiting was about to occur; the pulse was 120, and of good quality. The conjunctival reflex was present, the pupils equal and contracted. Thinking she was about to vomit, the head was turned to one side, and the ward sister was requested to keep an eye on her. About five minutes after this the house surgeon noticed that the pulse was 134, regular, and of good quality, that her respiration was unimpeded, though shallow, that she was cyanosed, and that the conjunctival reflex was present. He also thought she was about to vomit, and did not apprehend any special danger. Some ten minutes after the completion of the operation I again looked at her; she was then in the same condition. As I stood by her I noticed a sudden change: the blueness was suddenly replaced by a ghastly ashey grey colour. At that moment the sister drew my attention to the pulse, which had suddenly be-

come very bad, and after one or two sighing respirations breathing ceased. The head was at once lowered over the end of the stretcher, and artificial respiration by Sylvester's method commenced the tongue being drawn forward by forceps. Brandy and one twentieth grain of strychnine were injected into the upper arm, oxygen was given, and the interrupted current applied to the præcordium. So soon as artificial respiration had been fairly started it was ascertained that the pulse was absent both in the radial and femoral arteries, and no heart-sounds could be heard with the stethoscope. Artificial respiration was kept up for forty minutes, but at no time was any voluntary effort of respiration apparent. Whether pulse ceased first or the respiration I am unable to say, for the interval between the change of colour and the time when no pulse was found in the femoral artery was exceedingly short, certainly not more than a few seconds, and was employed in starting artificial respiration. During the performance of artificial respiration a large quantity,—I should estimate it as at least a pint,—of frothy watery secretion came away. I should like to emphasize the facts that at no time from the commencement of the administration up to the abandonment of artificial respiration was there any mechanical impediment to respiration in the upper air passages. and that up to the moment when the sudden change of colour was observed the pulse, though rapid, was of good quality. In all slightly over four ounces of ether had been used, which does not seem an excessive quantity for an administration lasting sixty-five minutes. A sample of the ether was handed to Mr. White, pharmacist to St. Thomas's Hospital, for analysis. He reports that it was of sp. gr. 720, and beyond a trace of alcohol contained no impurities. Ether from the same bottle had been used for other patients before and after the accident without any ill effects.

Report of post-mortem examination (made twenty-two hours after death by Dr. Turney).—Disease: abdominal exploration, acute œdema of lung following anæsthetic. M. G—, æt. 30, married. The body is that of a well-nourished woman; rigor mortis present. Abdomen: there is one surgical incision in the middle line between umbilicus and pubes, and another in the left iliac fossa. There is a little hæmorrhage beneath the

peritoneum in the left iliac fossa where it has been disturbed by the surgical manipulations, but otherwise the peritoneum is healthy so far as recent inflammation is concerned. In the pelvis there are signs of old adhesive peritonitis in the form of adhesions between the uterus and rectum. Both liver and kidneys are healthy, though engorged with blood. The spleen is small, but shows no signs of disease. The uterus is acutely retroverted from the dragging of the adhesions just mentioned. Its cavity contains some little blood-clot, as if menstruation were present, but is not otherwise altered. The left ovary is small, tough, and apparently atrophic, though it contains a recent corpus luteum. No signs of disease can be detected about either Fallopian tube. Both ureters and bladder are absolutely healthy. Thorax: both pleuræ are free from adhesions, and are healthy. The upper lobes of both lungs are slightly emphysematous; the lower lobe on the right side is tough, and contains very little air; the rest of both lungs are saturated with frothy fluid, which pours out of the bronchial tubes on pressure. The bronchi themselves are healthy. There are no signs of stomach contents in the air passages, and the fauces are clear. The heart is of normal size and proportions; the right side cavities are full of blood. [I may here remark that in the course of conversation Dr. Turney told me that though full of blood there appeared to be no distension of the right ventricle.] The muscle (heart muscle) appears healthy on section. All the valves are competent, and, barring an amount of atheroma rather unusual for the age of the body, are healthy. There is no sign of embolism or thrombosis in the pulmonary vessels. It should have been mentioned that neither stomach nor intestines show any deviation from the normal. The brain is engorged with blood, but is healthy; the pia is, however, distinctly milky in appearance, a condition, like the valvular atheroma, unusual in so young a woman.

As to the causation of death, it seems to me that the immediate cause was failure of the cardiac centre practically simultaneous with the failure of the respiratory centre. I myself believe that this failure was due to acute oxygen starvation; and as there was no impediment to the passage of a proper supply of air to and from the lungs, that the cause of this starvation must have lain in the

absence of a due interchange between the air and the blood in the alveoli of the lungs themselves, which may be explained by the condition of acute œdema of lungs found post mortem. In fact, that respiration failed, causing exhaustion of the cardiac centre. If this be so, then it is obvious that benefit from artificial respiration could scarcely be expected.

The PRESIDENT said it occurred to him to ask whether sections were made through the medulla oblongata. He remembered some years ago meeting with a case where acute œdema of the lungs followed upon a hæmorrhage taking place in the pons. That was a condition which one was fully alive to, and it was quite conceivable that hæmorrhage might have occurred in Mr. Grant Morris's case. Otherwise it was difficult to understand how an acute œdema arising from ether could have followed so rapidly as it appeared to have done in this case.

Dr. SILK said that Mr. Morris's case recalled very vividly to his mind what might be called an almost parallel case that occurred some little time ago at King's College Hospital. The patient was a very stout woman. As far as he could remember, a post-mortem examination could not be held, but undoubtedly there was œdema of the lungs. That such "water-logging" could be due to a central lesion seemed to him to be rather doubtful; he was more inclined to attribute the fatal result to the local action of an excessive supply of ether vapour upon the lung tissue itself. He was inclined to believe that the treatment by artificial respiration was of no service in such cases; indeed, he was disposed to think the proceeding might do harm. He did not try turning the patient on to the right side, but thought it might be the best line of treatment for such cases of acute ether poisoning. The other point about Mr. Morris's case was the albumin. They all knew that it had been a moot point whether ether should be given to patients who either had or had had albumin. It was not easy to say whether Mr. Morris's case proved one way or the other; still he thought such cases were on the border-line with those in which the administration of ether was of doubtful propriety, especially if the operation were a very long one.

Dr. STARLING said it occurred to him whether the condition which Mr. Morris described, and

the unfortunate ending, were due entirely to the administration of ether; whether they were not due, at any rate to some extent, to the condition of the kidneys. That was suggested to his mind by a case which occurred to him (Dr. Starling) a month or two ago. The patient was a lady aged forty-two, to whom he was to administer an anæsthetic for an exploratory operation on her kidney. Two years previously he had given her chloroform followed by ether, without any trouble whatever. At the later date she had been passing pus in her urine for five months, and the appointment was made for the surgeon to come down and explore her kidney; but two days previously to that he had a note from the doctor saying she was hardly in a fit condition for the anæsthetic, owing to apparent acute œdema of the lungs, as evidenced by quicker breathing, with some cyanosis and coughing, and the bringing up of a great quantity of frothy and clear fluid. She coughed up about fifteen ounces in the course of the day. When he (Dr. Starling) saw her the breathing was clear, and there was no trouble whatever. He anæsthetised her at the appointed time, giving chloroform with a Junker's inhaler instead of ether, as had been suggested at first. At one time, as she became rather cyanosed, he gave her a few breaths of A.C.E. mixture. She had no trouble, got well over the operation at the time, and had no vomiting afterwards. It occurred to him whether the condition of Mr. Morris's patient was due mainly to the condition of the kidneys rather than to the ether entirely.

Mr. DAVIS also made some remarks on this paper, and pointed out the possible dangers of giving much ether in cases of Bright's disease. Of late years he had preferred chloroform in such cases.

Mr. MORRIS, in reply to the President, said he was not present at the post-mortem examination, but he believed no hæmorrhage was found in the medulla, as ordinary sections were cut. He agreed with Dr. Silk that in a case of the kind no treatment of any kind would be of avail, because he had no doubt that at the time the patient ceased breathing she was already dead. He thought she died absolutely suddenly. Of course, directly he was sure she had gone wrong he took steps to bring about artificial respiration. It was probable that he misinterpreted the danger signal of shallow

respiration, but inasmuch as that was the usual preliminary to vomiting, he took it that this patient was preparing to vomit.

On the occurrence of Albumin and Jaundice after a brief administration of A.C.E. mixture.

Dr. SILK said that after the very elaborate notes which had just been read by Mr. Morris, he felt rather ashamed to come before the Society with a very fragmentary description of a case, but he held that it was one of the intentions of the Society for the members to place before one another particulars of any case which they thought to be abnormal. The case he proposed to record had, he thought, a bearing on Mr. Morris's. The patient was a woman aged sixty-five, in whom, after a brief administration of A.C.E. mixture, albumin and jaundice developed. That he thought was rather exceptional, and he did not know what was the explanation of it. She was admitted to King's College Hospital on January 18th, and was stout and over-fed, but otherwise in fairly good health. Nothing was found wrong with her urine; it was acid, sp. gr. 1010, no albumin, no sugar. She was in hospital eight days before an operation was performed, so that she had become accustomed to the changed conditions and surroundings, and during that time any jaundice or albumin would have been noticed. On January 26th she was placed on the table, and one of the clerks acting under him (Dr. Silk) anæsthetised her with A.C.E. mixture. The operation was merely one of incising the breast to see whether a lump in the breast was of a cancerous or innocent nature. As a matter of fact the breast was incised, looked at, and sewn up again, the whole procedure lasting barely fifteen minutes. During that time two ounces of A.C.E. mixture were placed on the sponge in the inhaler, so that there was enough to have lasted an additional fifteen minutes if it had been required; the anæsthesia passed off quietly and comfortably, and gave no anxiety. She was put to bed, and the next note, January 28th, was that albumin had appeared in her urine, but no sugar. On the 3rd of February the specific gravity of her urine was 1026, and albumin and bile were both present, and she was distinctly jaundiced; this condition continued until the 9th of February, when there was still a little albumin but no bile,

and but a faint tinge of jaundice. When she left the hospital, on February 11th, the albumin, bile, and jaundice had disappeared. The interest of the case consisted in the fact that it was possible to eliminate almost entirely the operative procedure as a cause of the condition, which must therefore be attributed to the anæsthetic. He would be glad to hear of any similar experience.

Miss ALDRICH-BLAKE said she had a case of a small boy aged eight or nine, who was the subject of hip disease, which was practically cured except for a small sinus. The urine was examined, but no albumin was found. It was of the ordinary specific gravity; she could not say exactly, as it happened two years ago, and she had not looked up the notes since. The operation consisted in scraping the sinus, and did not occupy much time. The anæsthetic used was chloroform. Twenty-four hours after that she was told that the patient was still vomiting. The urine was found then to be half solid with albumin after boiling, and it also contained some blood. This condition lasted about four days, after which he began to improve, and subsequently got quite well. He had previously had some suppuration, and probably the kidneys were affected by amyloid disease, but there was no albumin to be found until after the administration of the anæsthetic. There was no jaundice.

Dr. McCARDIE said that three weeks ago he administered ether to a woman who had had high temperature and some symptoms pointing to mischief near the kidney for a month previously. At the time of the operation there was a slight haze of albumin in the urine. On the morning following the operation there was a distinct increase in the albumin, which remained at the larger quantity for the next three or four days, and then gradually decreased to the former amount. At the operation an abscess in the cellular tissue about the kidney was found. No blood or other abnormal body was found in the urine.

The PRESIDENT said the great interest about Dr. Silk's case was the occurrence of jaundice associated with albuminuria following the giving of the mixture. He supposed it was a fact familiar to all, that any anæsthetic, particularly chloroform, did make people extremely bilious—they all knew the bilious vomiting and digestive derangements following the administration of anæsthetics, and

these conditions were earlier stages of what occurred, in Dr. Silk's case. He took it that in Dr. Silk's patient the jaundice was catarrhal. Probably the reason such cases were not more frequently met with was that Dr. Silk's patient probably was particularly inclined to catarrhal jaundice. No doubt many other cases had occurred but had been passed over because the observers had not such careful recorders as Dr. Silk. He had met with several instances of people being seriously upset after taking chloroform, jaundice being detected in the conjunctivæ in some cases of prolonged chloroform anæsthesiation. Perhaps one of the features of the greatest interest was the extremely small dose which would set up the hepatic irritation, and the complete absence of any possible interference caused by the operation. Some of the speakers had referred almost exclusively to the question of albuminuria following ether or following chloroform. Probably all the members of the Society were aware of the statistics which had been recently collected by various people, such as Dr. Campbell of Montreal. The sum total of these showed that albuminuria present before the anæsthetic was given was increased more by giving ether than by giving chloroform; while in the cases where there was no antecedent albuminuria, chloroform produced it in more cases than did ether. He believed that no statistics had yet been collected with regard to the effect of A.C.E.; and he did not think they really knew whether the A.C.E. acted *quâ* the ether or *quâ* the chloroform.

Mr. GRANT MORRIS said he had a case which bore not on the question of jaundice, but on albumin. It was a case of known kidney disease, and chloroform was selected under the impression that it was less likely to increase the damage to the kidney than ether. The sequel to the case was that the albumin was very much increased, and the amount of water was very much decreased; about the third day a complete suppression of urine set in, and the patient died. Off-hand he could not say how long the anæsthesia lasted, but he was sure of the other facts he had given.

(To be continued.)

NOTES, ETC.

The Abuse of Strychnine as a Stimulant.—

The papers which have been published in the medical journals during the last few years emphasising the value of strychnine as a cardiac and respiratory stimulant have been so numerous that many members of the medical profession are wont to regard it as being one of the best remedies which can be utilised to combat failure of either one of these vital functions. While we are thoroughly in accord with the view that strychnine is a powerful stimulant to these functions, we distinctly disagree with the habit which many practitioners have fallen into of employing this drug day after day for considerable periods of time for the purpose of stimulating the heart. Cases are frequently seen by us in which a physician has administered a dose of strychnine for a day or two with the result that there has been marked improvement in the symptoms. This improvement has then ceased to exist; and on the principle that the patient needed a larger dose a larger quantity has been given, until at the end of several weeks the patient has been taking quantities of this nervous stimulant and irritant which at the beginning would have been quite impossible.

As its use is continued, however, the improvement in the symptoms which first took place fails to be maintained, and finally, notwithstanding any dose that can be given, no relief is obtained from its use. In addition to the primary symptoms there is added a condition of excessive nervous irritability which in some patients is not only disagreeable but actually alarming, at least to the patient who has submitted to it, if not to the physician. Particularly is this true where very large doses of strychnine are administered to patients suffering from severe asthenic maladies, as for example in typhoid fever, tuberculosis, or epidemic influenza. In each and every one of these diseases a few doses of *nux vomica* or strychnine will frequently produce a noticeable improvement in the pulse and in the apparent condition of the nervous system, because strychnine being a powerful stimulant whips up the flagging nervous centres and causes them for the time being to perform their functions with greater activity. If the strychnine is persisted in, and ascending doses

are given for a considerable period of time, in addition to the nervous symptoms which we have mentioned, there is frequently developed an irritant fever, and particularly is this the case when strychnine is given in full doses during the later stages of typhoid fever or during convalescence from this disease, when, as is well known, anything which disturbs the nervous centres is very apt to result in a rise of temperature. Physicians are wont to watch the patient taking large doses of strychnine in order that this dose may be cut down as soon as twitching of muscles of the forearms or slight stiffness at the nape of the neck is developed, but in our experience, in asthenic patients, long before these symptoms appear there develops mental disquietude and a condition of what might be called "explosive nervousness," which is most unfortunate.

Only recently we have seen several cases which emphasise these points. One was a patient suffering from typhoid fever, who did not seem to take and use alcohol very well, although it was evident that his circulation required some stimulation. Strychnine was given to him in moderately large dose, with the result that for a day or two his circulation was improved, but at the end of that time the poor circulation returned and the patient began having evening rises of temperature of a considerable degree, which induced the physician in attendance to go back to the pure milk diet which had been previously rigorously enforced. As soon as the strychnine was taken away the fever ceased, and it became evident that there was no reason for modifying the patient's diet. In another instance a patient, suffering from the depression of influenza during convalescence, received large ascending doses of tincture of *nux vomica*, and became, as he expressed it, "wild with nervousness." In a third instance a student of medicine suffering from asthenopia was ordered, by a well-known ophthalmologist, ascending doses of *nux vomica*, which he took for a period of three weeks, at the end of which time he was taking thirty drops of the tincture three times a day. When he presented himself for treatment he complained of excessive nervousness, as he expressed it, "fearful thoughts," and suffered continually from a sensation that if he was not in a condition of activity something evil would happen to him.

These symptoms were entirely removed by stopping the *nux vomica*, and by directing the patient for a few days to take more than his usual quantity of outdoor exercise.

Strychnine or *nux vomica* is undoubtedly a remedy possessing great powers of stimulation, but we believe that its use should be limited to those cases in which we desire to call upon the nervous system for a sudden effort, as in meeting a crisis in a case of collapse or syncope. It is the lash of the whip which applied violently to a horse about to become "mired" may sting him into such an excess of activity that he raises himself from the quicksand in which he is sinking. So in medicine strychnine may be regarded as a valuable remedy to meet an emergency, but there is nothing which indicates that its continual use as a nervous or circulatory stimulant is wise, and there is much which indicates that such use of it is unwise.

Whatever influence strychnine may exercise upon the circulation is in reality due to its effect upon the nervous system, as it is not a direct cardiac stimulant of any considerable power.

In the discussion of a paper upon diphtheria, which recently took place in New York, Dr. Winters asserted that in cases of cardiac disturbance in diphtheria he had found that strychnine was practically useless, and he stated that Dr. Welch, of Philadelphia, entertained the same views. Personally, we agree with these gentlemen to the extent indicated by the previous remarks, but in sudden cardiac failure we believe that strychnine is the remedy which affords more hope of relief than any other which we can employ, although, as is well known, sudden cardiac failure in the course of diphtheria is one of the most fatal accidents which we have to combat.—Leading Article, *Therapeutic Gazette*, May, 1898.

The New Treatment of Posterior Spinal Sclerosis.—That a man "doesn't know what he can do till he tries" is well illustrated by the results obtained in Europe within the last two years in the treatment of organic ataxia, especially that of *tabes dorsalis* by the practice of systematic movements, concerning which an interesting article by Dr. J. Walsh, dated from Berlin, appears in the February number of the '*Therapeutic Gazette*.' The treatment consists essentially in a careful re-education

of the affected limbs by appropriate exercises, having for their aim the establishment of muscular co-ordination. That such a thing is possible is best proved by the fact that a considerable number of cases are now reported by eminent neurologists in which patients, bedridden in some cases for several years, have been put on their feet and enabled to regain the lost faculty to a serviceable degree. In such cases it is evident that sensation, while seriously obtunded, is not wholly lost; and the brain, through special education, is taught to discriminate and combine afferent impulses of much less than normal intensity. The treatment, which in many cases begins in bed, is at first confined to definite movements of the simplest kind, the object being the cultivation of precision. They are at first guided almost entirely by sight; later, when a certain degree of skill is attained, this guidance is dispensed with. In time the patient, having exercised his legs and feet in sitting posture, is put upright on crutches, and by carefully graduated exercises is taught step by step to walk; and, as his ability increases, these supports are substituted by a cane. As before stated, this method depends for its success upon the exactitude demanded for each separate movement. In addition, it is important to remember that the *séances* must, especially at first, be very brief, not averaging more than seven minutes twice a day. The intense attention needed in their execution involves a high tension in the cerebral motor centres, and undue strain results in great fatigue and discouragement. Contra-indications for this treatment are but few. Fulminating cases, with severe and constant pains, and marked bladder and rectal involvement, are not benefited. Those respond best in which the pathologic process seems to be in abeyance, while in the pre-ataxic stage the employment of this method has in a number of reported cases prevented in a marked degree the development of inco-ordination.—*Alienist and Neurologist*.

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NOTES FROM THE CLINIC OF W. ARBUTHNOT LANE, M.S.

On the Principles that should guide us in Operations.

IN the operative treatment of cancer our chief aim is the complete eradication of the disease, which should, if possible, be effected at all costs, for the reason that unless it is removed in its entirety it must in the ordinary course of events prove fatal. This should be made perfectly clear to the patient if any objection on the score of mechanical disability or deformity is urged against it. The extent of the operation must vary directly with that of the disease, and the more extensive and thorough the operative procedure for the removal of the growth the greater is the immediate risk to life from it. Of course, in a proportion of cases an operation is performed not so much with the object of removing the disease as of meeting complications consequent on its presence, as in intestinal obstruction due to growth. While the surgeon should be prepared to accept any risk that an extensive radical operation must involve, he should at the same time not omit to take infinite pains to render the condition of the patient such that a minimum of pain and discomfort shall result from his action. I think that we sometimes fail to bear this important point sufficiently in mind. I will illustrate my meaning by referring to certain operations on the larynx and tongue that have interested me. It was, I believe, the universal custom in excision of the larynx to leave the air passage in communication with the pharynx. While this state of affairs entailed of necessity upon the patient great pain and suffering, usually very much more than was experienced before the operation, it added very largely to the risks of surgical interference. It seemed to me that this method was clumsy and unscientific, and that the larynx could be removed quite as effectually and the discomfort and risk minimised by shutting off the communi-

cation between the air passage and pharynx. I applied this principle in an operation of excision of the larynx performed in February, 1891, upon a patient suffering from cancer of this organ, and in whom there were some infected glands which I feared could not be completely removed. The following is an extract from the report of the case in the 'British Medical Journal,' April 4th, 1891:—"It was determined to make the rest of his life as comfortable as possible, and to save him from any of the inconvenience which so frequently follows excision of the larynx from the passage of food, saliva, or serous discharge into the trachea, as well as from the risks of pulmonary complications which result from the same. For these reasons the mucous membrane, which had been separated from the back of the larynx, was carefully sutured to the hyoid bone and along the middle line, so that the pharynx was no longer in communication with the trachea. On the day following the operation he wrote on a slate that he had experienced no pain whatever, and that he was very comfortable. During the several succeeding days he appeared to suffer even less pain and discomfort than is entailed by an ordinary tracheotomy. He had little or no cough, and only expectorated a small quantity of mucus, the Trendelenburg cannula (which was inserted at the time of the operation and retained for some days) being removed and cleaned twice daily. Within forty-eight hours of the operation he was able to swallow milk with relish, and none of it escaped from the wound in his neck. A small leak formed later immediately below the hyoid bone, owing to his suddenly tearing off the dressing when in a passion. This closed quickly under treatment."

It is clear from this that the application of the principle of the separation of the air passage from the pharynx robbed of most of its terror an operation which was otherwise accompanied by so much pain, distress, and risk, and I am pleased to see that surgeons are now generally adopting it.

Again, in operations for the removal of the tongue the surgeon amputates this organ in a manner that differs very little in its crudeness from that which used to be adopted by the executioner in past times. A large raw surface is left which must heal in the most tedious manner possible by a process of granulation and cicatrisation. The abundant discharges from this surface, mixing with

the saliva are swallowed with difficulty, and afford a very good medium for the cultivation of all sorts of organisms. They are a source of danger, and are depreciating to the vitality of the patient. It is difficult to grasp the principle that leads the surgeon to amputate one portion of the body in this manner, while his mode of removal of other parts is influenced by a very different one. For instance, it would never occur to anyone in amputating a limb to leave a large raw surface of muscle unprovided with any skin covering to heal in this way if it were possible to avoid doing so. It cannot be that in making suitable flaps he is afraid of leaving some structure already affected by the disease, since this can easily be avoided in any case in which the tongue can be removed with reasonable hope of the complete eradication of the growth from the body.

Recognising how much the patient would be relieved from pain, distress, and risk by the substitution of a carefully devised flap operation for the gaping raw surface, and knowing the rapidity with which union takes place between such tissues in this region when they are brought into accurate apposition, I always adopt this method in my practice. The first recorded cases in which I operated in this manner are published in the 'Lancet,' 1892, p. 1291, in a paper entitled "A modification of the operation of partial or complete excision of the tongue," and the cases described in it were operated on in June and December, 1890. Somehow or another with a very few exceptions this mode of treating the stump of the tongue does not seem to have appealed to surgeons of the present day. Curiously enough, the late Dr. Widenham Mansell, one of the most original surgeons I have ever met, read a paper at Dunedin at the New Zealand Medical Association in 1890 entitled "A new method of excising the tongue," in which he advocated the use of flaps to close accurately the gap left after the removal of this organ. Mr. Alfred Parkin, of Hull, in a paper "On excision of the tongue," published in THE CLINICAL JOURNAL, August 25th, 1897, gives his experience of the application of this principle, which has proved most successful in his hands. I quote the following from his very interesting communication:—"Some surgeons might argue that small details of this description are, to say the least, unnecessary; to these one might say that

surgery ought to be (it often is anything but) the finest art, and that if I had to be operated on myself I would prefer a man who would, if it were possible, leave me half my tongue, who would prevent any blood whatever from trickling down my throat, and who would use all possible means to obtain a satisfactory healing of the resulting wound with the least risk of hæmorrhage or septic trouble, and with the least possible inconvenience to myself during the process."

Now as to the importance of effecting a thorough removal of diseased structures, it is too often the habit of the surgeon to rely on statistics in order to illustrate the merit of any particular operation. This would be all very well if the extent of the growth were the same in all cases or in several groups of cases, when various methods could be applied. Again, there is a factor that cannot be eliminated in practice, and that is the varying skill and cleanliness of different operators.

You must bear in mind that the wider the distribution of growth the more extensive must be the operative procedure for its eradication, and also the greater the probability of the reappearance, or, as it is commonly called, the recurrence of the disease.

Therefore it is absurd to argue against a big operation that it is more frequently followed by recurrence, and is accompanied by more immediate risks than a less extensive one, since the two operations are performed for quite different conditions.

Most surgeons will, I think, allow that the more thorough and careful are the operative measures for the removal of a primary malignant growth and of its secondary foci of infection, together with the channels along which such infection is carried, the more likely is the patient to escape its reappearance, though the immediate risks from the operation vary directly with its extent.

The surgeon should be guided in these operations by his sound common sense and by a knowledge of the behaviour of cancerous growths, and not attempt to follow slavishly the usually accepted teaching and methods, his procedure being modified by necessity by the wishes of the patient.

For instance, in amputation of the breast it is evident that the complete removal of the diseased organ with any affected tissues in the vicinity, the infected glands, and the channels along which in-

fection is carried, entails an extensive operative procedure, every step of which must be carried out skilfully, thoroughly, and patiently. I soon found that the removal of the infected lymphatic glands and channels could not be effected with anything approaching thoroughness through an incision made in the floor of the axilla. Consequently I adopted a method by means of which I was able to expose the entire axilla to its extreme upper limit, and to dissect out the whole of the fat with the lymphatic channels and glands contained in it. This I did by dividing the pectoral muscles and removing their fascial sheaths. If there was any suspicion of the muscles being involved they were cut away also.

Otherwise their divided ends were brought into apposition after the axilla had been cleared of its contents. If there was anything to lead one to suspect the presence of infection in the glands above the clavicle, this bone was drilled and then sawn across obliquely, so that part of the tract of the drill traversed either fragment. The sawn surfaces were forced apart, and the lymphatic structures beneath and above the clavicle thoroughly removed. This having been done, the clavicle was restored to its original form by means of a strong silver suture. The surgeon should spare no pains to excise every suspicious structure, and he should never rely on his finger alone to decide on the presence of secondary infection, since it is clear that it can be no real test; besides, delicacy of touch varies within wide limits in different people. He should see everything, and be inclined to regard any doubtful structure with the greatest suspicion, knowing that the removal of a healthy lymphatic gland can do little harm, while the presence of a residual focus of infection may nullify the results of a very extensive operation. It is entirely owing to a want of thoroughness on the part of earlier operators that the surgical procedures for cancer of the breast obtained a very bad name, being regarded with well-merited suspicion not only by the public, but also by the profession. This reproach can only be removed by the adoption of improved and more efficient methods.

I described the measures referred to above in a paper published in the 'Transactions' of the Clinical Society, 1893, entitled "A case illustrating a more effectual method of removing a cancerous breast,

lymphatics, and glands," and gave my reasons for the course pursued.

Perhaps the most serious difficulty which one experiences in operations on the breast is the provision of a satisfactory skin covering when there has been extensive skin infiltration. In most cases this can be met more or less satisfactorily by the formation of suitable flaps or by grafts; but there are others in which the surgeon must either hold his hand or adopt some more efficient means of obtaining a cutaneous covering. As an illustration of this difficulty and of the manner in which it can be met, I will quote the following from a short paper published in the '*Lancet*,' October 12th, 1895, entitled "A Case illustrating a very effective method of treating extensive malignant disease of the breast."

One comes across cases, however, in which the skin is so extensively infected that this last operation is unsatisfactory owing to the large raw surface which is necessarily left. In such cases I would urge what at first sight appears to be a heroic measure, namely, amputation of the arm at the shoulder in order to obtain enough skin to cover the raw surface and to give a nice soft covering to the part. The patient will certainly die if she is not operated on, and in all probability the arm will shortly become a source of distress to the sufferer owing to interference with the venous channels; therefore it is no great deprivation to remove it. Besides, it diminishes the risk of the operation, it ensures immediate union, and within a few days the patient is enabled to get about and enjoy an active existence. I cannot do better than illustrate the method I here advocate by the report of a case.

A woman aged twenty-nine years, single, of a remarkably youthful appearance, was admitted under my care into Guy's Hospital on September 9th, 1895, suffering from carcinoma of the right breast, which she had observed for the first time five months before. The mass was large, and involved almost the entire breast. The skin was adherent over it, and was ulcerated over a space as large as a five-shilling piece. All about the ulcer and for an area around the breast, the skin presented minute nodules of infection. There were enlarged glands in the axilla, and in the subcutaneous tissue over the coracoid process a gland could be felt. It had apparently been infected

from some cutaneous nodules just below it. Nothing could be felt in the liver, and the patient's general health and weight were as good as ever they were. The advantage of removing the arm, the possible benefit she would derive from it, and the risks she ran were fully explained to her, and she insisted on undergoing the operation. An incision was made along the length of the clavicle

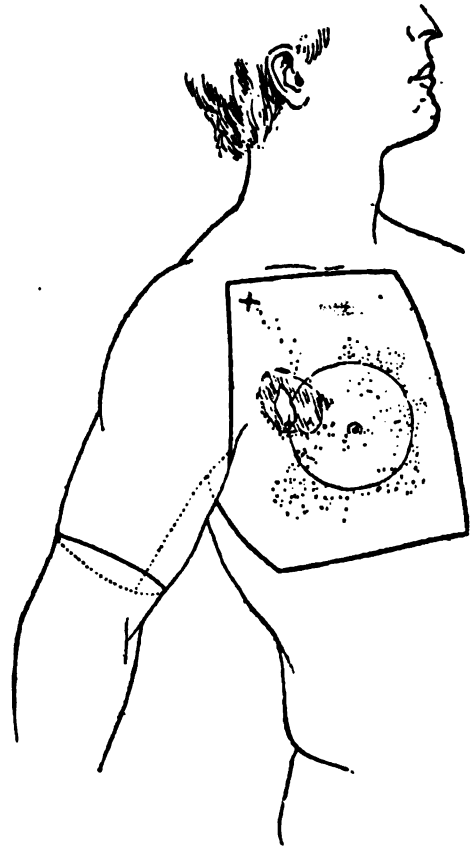


Fig. 1.—The above illustration shows the positions of the incisions, with the ulcerated surface in the right and upper part of the breast. The \times indicates the position of the gland in the subcutaneous tissue, and the dots show the approximate distribution of the points of infection of the skin by growth.

to the middle line, and the middle third or more of the clavicle was exposed and removed. The subclavian artery and vein were tied at the margin of the scalenus anticus, and all the areolar tissue and glands in the subclavian triangle carefully dissected out. There was no obvious infection of these glands, but beneath the clavicle the upper limit of the enlarged axillary glands was defined,

and the glands were turned downwards. Great care was taken not to injure the supra-scapular or other vessels crossing the subclavian triangle. From the inner limit of the horizontal incision another was carried down vertically along the middle line for about nine inches. It lay well outside the limit of skin infection. From its lower end another incision was carried backwards to the posterior fold of the axilla, at what seemed a safe distance from the growth, and then it was run upwards along the posterior fold to the axilla, when it ran across the arm to the outer end of the clavicle. A vertical incision was made from the upper limit of the posterior axillary fold to the elbow, where it was crossed by a circular incision. The skin and subcutaneous tissue were dissected up off the deep fascia as far as the deltoid, out of which a large and sufficient flap was cut to ensure as much vascularity of the flap as possible. The inner part of the deltoid was removed, and the limb disarticulated. The skin and all the subjacent soft parts were dissected off the sternum, clavicle, ribs, cartilages, intercostal muscles, serratus magnus, and posterior wall of the axilla, and the great square flap, which measured more than nine inches along each side, was fitted accurately and comfortably into the large exposed raw surface. The part that corresponded to the circular incision around the arm was united to the edge of the median or sternal incision, while the edges of the vertical incision in the arm were connected to the upper and lower horizontal incisions in the thorax. Very little blood was lost during the operation. The growth was found to involve the greater pectoral, but the most careful subsequent examination of the parts excised showed that the primary growth, the skin, and glands were very freely removed. The patient ran the usual course of an aseptic wound, experiencing no discomfort or pain in the part. The illustration represents diagrammatically the condition of the patient before the operation, and it shows very well the outline of the skin incisions in the chest and arm. Of course the area of the surface covered by the skin of the arm, after healing had taken place, was considerably smaller than when the operation was done, owing to a certain amount of shrinkage of the loose flap.

I would just point out that the removal of a considerable portion of the clavicle enabled me

not only to remove all glandular and areolar tissue beneath and about it, and to tie the vessels securely, and with a minimum loss of time, but it also allowed the scapula to fall into such a position as to give a greater length of flap, and a less strain upon it than if the clavicle were left intact. Every operating surgeon of large experience is confronted too often with the results of very imperfect removal of the diseased structures, so much so that he sometimes cannot but think that the operator was influenced by a desire not so much to effect the thorough removal of the affected parts as to obtain a rapid recovery. It would seem obvious that such a course is not likely to reflect much credit on surgeons, and especially on operations for cancer of the breast.

In the last condition to which I will call your attention the question of subsequent comfort and effectual removal are both involved. I refer to cancer of that portion of the rectum that is covered by peritoneum, and which does not lend itself readily to resection in the majority of cases. In many of such cases surgeons appear to have obtained excellent results by the operative procedure which is commonly described as Kraske's operation. I am sorry to say that I have not been able to benefit my patients as much as I would like by the use of this method, and for the following reasons:

Firstly, I was rarely able to remove the infected glands in the mesentery with any feeling of certainty.

Secondly, if much bone was removed the discomfort experienced by the patient after the operation was often great and prolonged.

In my experience these cases are as a rule best treated by the performance of an inguinal colotomy,

asc 2

Fig. 2.

and the aperture fitted by a cup valve plug as made for me by Down Bros., which gives the patient complete control (see Fig. 2). This having been done, an effort should be made to remove not only

the primary growth, but also the mesentery with the infected glands in it.

The varying conditions presented by these growths, by the secondary infections in the mesentery, and the different lengths of the meso-sigmoid, must influence the details of the procedure considerably, but the principle underlying our action must be the same in all cases, namely, the complete eradication of the primary growth, and of the infected glands. Perhaps I can best illustrate the methods I adopt by describing a case.

A woman aged fifty-six was admitted into Guy's Hospital on 27th April, 1891, under Dr. Perry, suffering from acute intestinal obstruction. She was in a very prostrate condition. A small hard growth could be felt high up in the rectum. The upper limit of the sigmoid was brought to the surface immediately above the internal abdominal ring, and an aperture made in it. Before this was done the pelvis was carefully examined, when the growth was found to involve the middle third of the rectum, in the mesentery of which large glands could be felt. The condition of the patient precluded any further interference at the time. After she had sufficiently regained her strength to bear a more prolonged operation I opened the abdomen in the middle line, with three methods in my mind by means of which I hoped to effect the complete removal of the growth.

One was to excise the growth and establish continuity by means of Murphy's buttons, which I have always found most satisfactory since I used them in January, 1894, the first time they were used in England ('Lancet,' 1894, p. 1006). The second was to remove the whole or most of the bowel beyond the opening of the sigmoid in the groin; while the third was to excise the growth with its mesentery, together with the portion of the rectum below it, and to bring the lower end of the sigmoid, or rather its junction with the rectum, down to the anus.

The result of a careful examination of the mesentery of the rectum and sigmoid decided me on taking the third course, feeling that if I failed in it I could always fall back on the second.

The rectum and mesentery were separated from the sacrum, the mesenteric vessels being tied. The peritoneum of the meso-sigmoid was also divided in such a manner as to allow of its elongation. The patient was placed in the lithotomy

position, when an incision was made around the anus, which was then sewn up. An incision being made backwards to the coccyx the rectum was dissected from the vagina and sacrum. It was then possible to draw the whole of the rectum out through the wound in front of the coccyx. Owing to the vessels having been tied in the mesentery this portion of the operation was accompanied by very little loss of blood. The bowel and growth were removed, and the part above the growth fixed in the wound. The patient made an uninterrupted recovery. Some of the mucous membrane of the attached bowel sloughed, but it soon separated. All the fecal matter escaped through the hole in the groin. The sinus in front of the coccyx gave her no discomfort whatever. It seems to me that by this method we applied our two principles as effectually as possible.

On the Treatment of Ununited Fractures.

Perhaps few operations in surgery may make a greater demand on the manipulative dexterity and skill of the surgeon than an ununited fracture of the tibia in its upper third, especially when the direction taken by the new joint is somewhat irregular and oblique in two planes, as is so often the case in this situation.

In such operations, after the removal of the surfaces of bone with the dense fibrous tissue connecting them, the ends of the tibia are separated by a considerable interval. The chief difficulty in this operation consists in determining the plane in which the fracture lies, and in cutting away the false joint so as to leave the tibial surfaces perfectly even and parallel to one another.

To allow of the approximation of the tibial surfaces a corresponding length of the fibula must be removed, and if there exists also an ununited fracture of that bone it should be excised. If there is no ununited fracture of the fibula a length of it is exposed and a piece equal to the interval between the tibial surfaces is removed by parallel oblique sections. It is much more difficult than would appear at first sight to gauge accurately the exact length of the fibula that should be cut out, and one is very much inclined to take away too much than too little. This is a matter of little importance, since should too much be removed, after the tibial surfaces have been united a portion of the

fragment can be cut off and threaded on the wire, uniting the two ends of the fibula.

Even after the fibula has been shortened the soft parts about the gap in the tibia, and to a lesser degree about that in the fibula, offer a considerable resistance to the approximation of the tibial surfaces, which can only be secured permanently by the use of steel screws. Wire alone is of very little service in this situation. Indeed, I might say that in operations for ununited fracture in any situation I always use one screw at least to overcome the separating force which is brought about by the consequent buckling of the soft parts,—a force exerted in an opposite direction to that in a recent fracture,—and to secure the most accurate apposition and immobility of the fragments upon one another. In none of the numerous cases in which I have interfered in this way have I failed to obtain union, and some few of these had been operated on unsuccessfully by others once and sometimes twice previously.

Fig. 3, which represents a radiograph taken for me by Messrs. W. Watson and Sons, is an excellent illustration of the result of the operative procedure in such a fracture. The man had broken his leg more than a year before. At the time he came under my care the fragments moved freely on one another on an axis at right angles to the plane of the fracture, which was very oblique in direction, and the patient was altogether unable to bear his weight on the limb. In this case it was necessary to use three long steel screws passed in different directions in order to secure perfect apposition and complete immobility of the fragments. Two loops of silver wire were found lying loose in the bone, and afforded the only evidence of a previous operation. A little too much of the fibula was removed, and, as is seen in the illustration, a portion of it was replaced. The radiograph was taken about three months after the operation, the result of which was perfect, the patient being able to bear his weight securely on the damaged leg.

CASE 2.—A woman about 40 years old sustained a fracture of the tibia and fibula thirteen months before she came under my care. A radiograph showed that the tibia was fractured at the junction of the upper two thirds with the lower third of the bone, the upper fragment lying in front and to the inner side of the lower. The fibula was broken in two places. The fragments of the tibia moved

upon one another, but the fibular fractures had apparently united firmly. She was quite unable to stand on the broken leg. On exposing the tibial fracture the ends of the bone were found to be united by a more or less calcified fibrous tissue. The necessary portion of bone to be removed was carefully defined, and after it was sawn off the surfaces could be brought into accurate apposition by the lion forceps. By means of a steel screw and a couple of loops of

Fig. 3

silver wire the tibial fragments were secured immovably together. The bones united firmly.

CASE 3 was a woman about 35 years of age, who about fifteen months before broke her femur at the junction of the upper fourth with the lower three fourths of the shaft. She had never been able to bear her weight on the limb since. The upper fragment projected forwards and outwards, overlapping the lower, which lay behind it and to its inner side. The bones moved freely on one another around an axis which passed through both fragments. The ends of the bone were exposed.

A dense mass of imperfectly calcified fibrous tissue united the lower end of the upper fragment with the outer aspect of the lower, the opposing overlapping surfaces being covered by a serous surface in the sac of which was a quantity of glairy fluid. With some difficulty the surfaces of the fragments were removed, and the exposed ends of the femur brought accurately and securely together by two strong steel screws. The patient is doing exceedingly well; the operation, which necessarily involved very extensive incisions, being followed by practically no rise of temperature.

CASE 4 was a man about 40, who sustained a fracture of the humerus at the junction of the middle with the lower third more than a year before. The fragments did not unite by bone, a flail-like limb resulting. He had undergone two operations in the meantime, the mobility of the fragments being, however, unaltered. He was sent into the hospital for amputation. On cutting down on the false joint a couple of loops of silver wire were found lying loose in the bone. These were removed, and the ununited fracture cut away by sawing through the bone obliquely above and below it. A single screw sufficed to retain the fragments securely in accurate apposition. The bone united perfectly.

I might enumerate many more examples of this operation, but these suffice for the purpose. As a matter of fact, the excellent radiograph which illustrates the first case forms my only excuse for inflicting these remarks on you, since I discussed the advantages of this treatment fully in the 'Lancet,' December, 1893, in a paper entitled "On the advantages of the steel screw in the treatment of ununited fractures."

Case of Death following Forced Reduction in Pott's Disease.—A boy of twelve, who had had Pott's Disease for eight years, was treated by Callo's method of forced extension. The kyphos was in the dorso-lumbar region. After the operation epistaxis and difficult respiration were noticed, but on the eleventh day the dyspnoea became augmented and death occurred. At the autopsy a fibrinous pleurisy with hæmorrhagic fluid in the right pleural sac was found, together with softening of the fragments of the diseased vertebræ, and a rupture in the anterior wall of the tuberculous abscess.—*Archives of Pediatrics*, June, 1898.

CHAPTERS FROM THE TEACHING OF DR. G. V. POORE.

No. I.

GENTLEMEN, — Medical jurisprudence must be defined as the application of medical knowledge to legal purposes. It is sometimes spoken of as forensic medicine—that is to say, the medicine of the forum or public place. It is a branch of state medicine; the other branches being hygiene and public health.

Cases involving knowledge of medical jurisprudence are of everyday occurrence, such as sudden death, assault, homicide, murder, poisoning, and damages for injuries, questions of identity, legitimacy, and insanity, and questions in relation to life assurance and benefit societies.

It is needless to point out to you the importance of the study of medical jurisprudence. There is no escape from it, not by any possibility. A man is free to choose his own line of practice; he may be a general practitioner, a surgeon, an obstetrician, a physician, oculist, dentist, or anything you like; but medical jurisprudence is very often a thing which we run into willy nilly. A man is knocked down in the street—you happen to be the nearest person having professional knowledge, or you are called out to a serious accident which happens in the neighbourhood of your house. You cannot refuse to go; common humanity compels you to go, whatever your practice may be. But you have to follow that case into the law courts. It is important to remember that, whereas the practice of medicine is usually confidential, in courts of law no concealment is possible, and your conduct with regard to the case in question, whether it be wise or whether it be foolish, is canvassed by the public at large. A reputation may be either made or marred according as your conduct in the witness-box shows that you have behaved sensibly or foolishly in relation to some *cause célèbre*.

Now in giving a course of lectures on medical jurisprudence, it is not my intention to talk text-book at you; and I wish to warn you that my lectures will not replace a text-book; they are to be an auxiliary to the text-book, and will help you, I hope, in reading your text-books. There could

be no object in a lecturer giving dry figures and tabular statements, and so forth, which are much better printed than uttered; and it is not my intention to do anything of that kind. But I hope to be able to interest you, and to show you that in the lecture room it is possible by a little freedom of treatment, to give you, not only the bricks with which you will build the edifice of your knowledge of medical jurisprudence, but a little mortar also in the form of interesting details which will aid your memories.

Medical jurisprudence is a comparatively modern science. There always must have been cases in which medical jurisprudence was necessary, but it was long before the necessary knowledge was systematised.

The first writer on medical jurisprudence was undoubtedly Zacchias, who was physician to Pope Innocent X. He wrote a large book called '*Quæstiones Medico-legales*,' in which he discussed many of the questions which we shall discuss. Zacchias was one of the household of the Pope; he was a priest as well as a physician, and in his great book there is a strange and interesting intermingling of questions of medicine with questions of theology.

The first writer in this country was Dr. Farr, who wrote in 1788. One of the first French writers was Foderé, who wrote in 1796. The founder of modern toxicology was Orfila, a Spaniard domiciled in France. Orfila's book on poisons is certainly the foundation of the modern science of toxicology. I do not know when the first edition of Orfila's book was published, but I have a copy of the second edition, which was published in this country in 1819. A notable English treatise on medical jurisprudence was that by Paris and Fonblanque. Dr. Paris was president of the College of Physicians, and Mr. Fonblanque was an eminent barrister; they combined, and produced a most excellent book.

Then we come to the great work on toxicology by Sir Robert Christison, based largely on the previous work of Orfila. Next in order is Alfred Swain Taylor, whose '*Medical Jurisprudence*' is a classic, and always will remain so; it is a magnificent book, and in it you will find excellent summaries of many of the great trials. '*Taylor's Medical Jurisprudence*,' as edited by Dr. Stevenson, of Guy's Hospital, still retains its high character.

In selecting a text-book for medical jurisprudence you have a large choice. Most of you do not want a very big book: you want facts. I can only say that the works edited by Dr. Stevenson, especially the small "*Taylor*," as it is called, with the works of Dr. Dixon Mann, Dr. Luff, and Guy are all good, and I believe perfectly reliable. But they are a little short, and most people, I think, find it difficult to carry in the mind facts which are stated too succinctly, and I strongly advise you to get one of the bigger books in addition; and you must be strangely constituted if you do not find a great deal of interest in it.

The knowledge required by a medical jurist is very wide. There are in this country no specialists in medical jurisprudence, for the very good reason that there is not a living to be made out of it. In this country we do things in a haphazard way, but the results are nevertheless fairly good. The investigation of cases of poisoning, cases of assault, and so forth is undertaken in the first instance always by the practitioner who happens to be in attendance on the case, and as a rule that works fairly well. Our modes of procedure in cases involving medical jurisprudence differ from the Continental modes. On that account foreign books on medical jurisprudence are not so useful to an Englishman as the English books. Of course, on questions of pathology and pure chemistry you may get a great deal of very valuable information from foreign sources, but the whole management of a case involving medical jurisprudence is different abroad to what it is in this country. I have said that the knowledge required of you is very wide, and that is one of the advantages of medical jurisprudence; it compels you to attend to a good many questions. But the study of it is of great value in itself. In medicine and in surgery, but more especially in medicine, we are always confronted with a plurality of causes. A man comes before us with, say, pneumonia, and we ask what is the cause of it? He has, perhaps, been in a house where there are several cases of pneumonia, he may have been chilled, he may have got diseased kidneys, he may have been starved, he may be a drunkard, he may have been injured, and out of the many causes you are often puzzled to say which have been the exciting, predisposing, and determining causes. While in medicine we have the plurality of causes and the singularity of

effect, in medical jurisprudence we have the other condition of things—the singularity of cause and the plurality of effects. And it is a very interesting thing to see how many effects may follow a single cause. When we discuss toxicology you will see the truth of this.

One of the chief duties you will have to perform as medical jurists—and you are all bound to be medical jurists—is to give evidence. You may have to give evidence in the sanded parlour of a public-house at a coroner's inquest; and you must remember that evidence, given possibly under squalid and hurried circumstances, may lead you to a magistrate's court or to the assize court. You must remember, therefore, that whenever you open your mouth as a medical jurist you have got very carefully to consider your utterances. Never give evidence without thoroughly considering it and without recognising the importance of it.

Evidence is of many kinds. First of all, it may be *oral* or it may be *documentary*. In this country almost all evidence is oral—given by word of mouth. Again, evidence is either *direct* or *circumstantial*. What is the exact meaning of that? Let us suppose there is a murder committed in Gower Street by A. If I say I saw A stab B in Gower Street at ten o'clock, that is direct evidence. If I say I saw A going down University Street towards Gower Street with a dagger in his hand at two minutes to ten, that is a circumstance which may lead to A's conviction. It has been debated whether circumstantial evidence or direct evidence is the more valuable, and it is very difficult to say. I say that I saw A going down Gower Street, but, with the best intention of speaking the truth, I may be mistaken—I may have mistaken the identity of A. If a series of persons give a series of circumstances all implicating A, then circumstantial evidence is quite as valuable as direct evidence. Circumstances, if you get a complete chain of them, are very valuable indeed, but the chain ought to be complete. It has been said that circumstances cannot lie. True, circumstances cannot lie, but of course there must always be a doubt as to whether the witnesses are credible or not.

Next, the evidence you give may be either *common* or *skilled*. I may put it in this way: If I say I saw A stab B in Gower Street, I may be subpoenaed by the police as a common witness; then I go to testify to facts, and it is my duty as a citizen

to do so. But if the police come to me and say, "A man has been found dead in Gower Street, with certain wounds upon him, and we want you to say whether these wounds are the cause of death," then I go as a skilled witness to give opinions based on facts, and not to testify to facts which I have seen myself.

The evidence which you give in court must be relevant. Your evidence must have reference to the facts which are under discussion in the court. The judge is the only decider as to relevancy, and you very often find that barristers try to shake the credibility of the witness by putting awkward questions as to character and so forth, the relevancy of which is very doubtful. You are not bound to answer any question which may incriminate yourself. In the old days of duelling any doctor who went out as surgeon to a duel became an accessory to the fact if the duel resulted in death. Technically he became accessory to a murder. Therefore in such a case if the doctor was asked whether he went, knowing that a duel was to be fought, he was not bound to answer that question because the answer to it might incriminate himself.

It is important to remember that *hearsay* is *not* evidence. Evidence must be first hand, and you must be very careful to distinguish between hearsay and evidence. C may go into the witness-box and say that he saw a man killed in Gower Street, but B cannot say that C told him he saw a man murdered in Gower Street. A very important decision under this head occurred some years ago at the Norwich assizes, and I will read it to you.

"*Regina v. Bedingfield*, Norwich Assizes, November 13th, 1879, before Cockburn, C. J. Henry Bedingfield was tried for the murder of Eliza Rudd at Ipswich on July 8th, 1879. Bedingfield and Rudd were on most intimate terms, and had been known to quarrel at times. They were heard to quarrel on the morning of July 8th. About eight o'clock Rudd was heard to scream, and was seen to run from the house with her throat cut. She was taken back into the house, where she died in ten minutes, and she pointed to the next room, where Bedingfield was found with his throat also cut, lying upon his belly with his left arm under his head, with his right arm extended, and near the right hand an open razor. The right hand and razor were both bloody. The wound in Rudd's throat was three

or four inches long, sloping upwards from left to right, and deeper towards the right. It divided the oesophagus and big vessels. The wound in Beddingfield's throat was transverse, above the thyroid cartilage, and both sides alike. The defence set up by the prisoner was that Rudd had first cut his throat and then cut her own. He was convicted and sentenced to death.

Rudd when she rushed from the house screaming and with her throat cut, made a statement to one of the witnesses. The statement probably incriminated the prisoner, but it was not admitted as evidence because it was made after the deed and not in the presence of the prisoner. It was hearsay, and not part of the *res gestæ*. It was not admitted as a dying declaration because there was no evidence that the woman knew she was dying.

That is perhaps the strictest decision ever made with regard to hearsay and a dying declaration. This was the decision of Sir Alexander Cockburn, one of the ablest judges who ever sat on the bench. It has been disputed by Mr. Pitt Taylor and others. This decision also presses upon us the fact that dying declarations are not evidence unless the person making the declaration knows that death is imminent. The decision as to whether a dying declaration is to be accepted as evidence or not rests solely with the judge, but in order that a dying declaration may be accepted you must be able to state that you are perfectly sure that the person who made it knew that death was imminent.

When you are called upon to give evidence about a case, consider it from every point; and you would do well to consult any books you may have. You will also do well to talk over the matter with a discreet friend. Before you give your evidence you must try and look at the case from all points of view. Remember that you are going to give evidence which may incriminate A or B, and you must consider what A or B might urge in his defence. In that way you will weigh the question from both standpoints. Having once made up your mind, give your evidence, and do not trouble about the consequences.

Do not form a judgment on insufficient data; that is the first thing. In times past very much oftener than now one used to hear of coroner's juries returning a verdict of "death by the visitation of God." Well, every death is by the visitation

of God, and if coroner's juries are only to tell us that, they are of no use. The coroner's jury has nothing to do with ultimate causes; it is only concerned with proximate causes, concerning which you cannot form a conclusion without data. If the data are not forthcoming, do not give evidence. On the other hand, do not be foolishly obstinate. A man is knocked down in the street, and he is obviously injured, and the injuries which you can see are quite sufficient to account for his death; do not insist upon a needless post-mortem examination—that would be foolish. You may urge that it will be advisable, but it is notorious that in many cases of sudden death the cause of the death is practically certain without a post-mortem. If a man drops suddenly dead, and you know that he has got or had aortic disease, do not insist on a post-mortem examination. You may say "he had a disease which in my judgment was quite likely to cause sudden death." If death has nevertheless really been caused by a dose of poison, that is not your fault, but you would be quite right in giving the above opinion. Post-mortems are of course always interesting and instructive, but that is another matter. In your public capacity do not needlessly insist upon them.

In a court your examination, as a rule, is in three parts. First there is the *examination in chief*. If you are called by the prosecution, the counsel for the prosecution elicits your evidence. He probably knows from previous depositions exactly what you are going to say, and he elicits your evidence by questions in this way:—You are Dr. Smith? Yes. You are a doctor of medicine? Yes. You are practising in such and such a place? Yes. You saw the deceased? Yes. He had his throat cut? Yes. And so on. That is the examination in chief. In giving evidence be simple, and be careful of the language you use. Unfortunately medicine is hedged about by very long words, and the use of jargon has become a mischievous habit with most of us. It is a very important thing indeed when you are in the witness-box not to use puzzling terms. When I was house surgeon over the way I had to give evidence on one occasion in a case of injury, in which a man had been knocked down by an engine. I made use of two expressions: I described the various wounds the man had, and amongst others one going from the *commissure* of

the eyelids backwards, and I said that after he was admitted he suffered from *traumatic* delirium. The newspapers next day reported that the man was a "connoisseur of the eyelids," and had "aromatic delirium."

Taylor reports a case where a medical witness informed the court "that on examining the prosecutor he found him suffering from a severe contusion of the integuments under the left orbit, with great extravasation of blood and ecchymosis in the surrounding cellular tissue, which was in a tumified state. There was also considerable abrasion of the cuticle."

Judge.—You mean, I suppose, that the man had a black eye?

Witness.—Yes.

Judge.—Then why don't you say so at once?

I bring forward these cases because it is very annoying indeed to find that in the newspaper reports of trials and so forth you are down as having talked rubbish; and you will be sure to be reported as having talked rubbish unless you are exceedingly careful to explain what you mean. Medical jargon is sometimes only a screen for ignorance. It is said that everybody will talk sensibly and well about a thing he really understands, and I think that is true. It is also said that children of moderate age make excellent witnesses, because they are perfectly simple. You cannot shake them; they are guileless, they have no thought of traps being laid for them in subsequent cross-examination, and so forth.

In the witness-box be dry; avoid adjectives and adverbs and metaphorical expressions. Say that the wound was so many inches long; that the man may have lost six ounces of blood. It is much better than saying "he had a frightful wound" and "enormous hæmorrhage," or words to that effect. Be dry, and let your statement be as accurate as possible, because if you use expressions which are not accurate, such as "enormous" and "frightful," you are sure to be cross-examined upon them; and a clever barrister, if he does not shake your evidence, may have the satisfaction of making you appear a little ridiculous before the jury, and that may serve his purpose very well.

The next point is with regard to notes. You go to a case and you make notes of the circumstances, and the question is, May you use those notes in court? I answer, Yes and No. You may use

your notes to refresh your memory. For instance, you may be asked, "Now, was it Monday or Tuesday you were at such and such a place?" You may then take out your pocket-book and refer to it. The judge will let you do it, but you must be ready to swear that the notes made in your pocket-book were made at the time; that they are not "cooked" notes—notes which you have concocted after consideration. That is very important and quite reasonable, and they must be used only to refresh your memory, not to read from as evidence.

Next with regard to professional secrets. You have no right to speak of what passes in the sick-room to anybody except in the witness-box; then you are bound to, and the judge will tell you that you are bound to. But I give you this piece of advice: never utter professional secrets without appealing to the judge. You know perfectly well what the result will be—you will have to tell them. But I think it is a wise course to make it appear to the public that you divulge these professional secrets under judicial compulsion. I have taken that course more than once myself.

I remember one case of a report made in an insurance case. It was a report which rather called in question the character of a man for temperance. It was possibly a libellous statement, which was made confidentially. I said, "This is a confidential document made to the directors, and to them only." The judge said, "You must state it," as I expected he would; but I made it appear that the evidence was given only under judicial compulsion. The priest in Roman Catholic countries is allowed to escape from this compulsion. What is said to him under seal of confession need not be divulged.

After your examination-in-chief you are cross-examined, and it is, of course, with a view to shaking your testimony. Whereas in the examination-in-chief only simple questions are allowed, in the cross-examination "leading" questions are permitted. What is the difference? "Where were you at ten o'clock on Tuesday?" "I was in Gower Street." That is a simple question. A cross-examining barrister can say, "Were you in Gower Street at ten o'clock on Tuesday?" That is a leading question, and suggests the answer. If a leading question is put in examination-in-chief, the opposing barrister will say, "I object to that question."

MEETING OF THE SOCIETY OF ANÆSTHETISTS.

At 20, Hanover Square, April 21st, 1898.

The President, Dr. DUDLEY BUXTON,
in the Chair.

(Continued from p. 138.)

DR. STARLING read the following communication from Mr. Chapman on a death from the administration of chloroform.

MR. PRESIDENT AND GENTLEMEN,—At the invitation of your secretary, Dr. Starling, I have to report a case of syncope early in chloroform administration.

The patient, a healthy woman of 35, was admitted into hospital for removal of sarcomata of arm; the usual preparation was carried out, and the patient walked into the theatre. I proceeded to administer chloroform in my ordinary method on a folded piece of lint pinched into shape by the fingers, and immediately after dropping the first amount of chloroform on to the lint (about twelve minims) the patient, who had been breathing in rather an agitated manner, held her breath, but upon being reassured took three or four inspirations and held it again; during this time the pulse had been gradually losing its tension—a phenomenon which in some 1000 cases I have noticed to be always present in varying degrees—till it was hardly perceptible. I immediately removed the lint from her face, and at the same time lost all feel of the pulse; the face was then grey-white and rigid, with pupils widely dilated, the whole aspect being that of extreme terror. I pulled the tongue sharply forward, and the patient made one or two convulsive heaves of the chest and abdomen; artificial respiration was commenced and maintained by the visiting surgeons present, and the legs and body were elevated for a short time, ether and strychnine were injected, flapping with towels, faradism, and venesection were also tried, but without avail; a method which some years ago I found successful with a colleague's case, viz. rhythmical and forcible compression of the heart, was not tried till very late.

At the post-mortem examination the heart was

found quite empty and lax, slightly below normal weight but otherwise healthy; the veins throughout the body were gorged with blood, more particularly the abdominal ones; the lungs slightly congested, but the brain anæmic. All the other organs were quite normal, and there was a free air-way.

This case, occurring just before the publication last year in the 'British Medical Journal' of a paper read by Dr. Leonard Hill before this Society, I think bears out some of the theories and conclusions then expressed: the very early termination, the initial voluntary stoppage of respiration followed by lowered arterial tension, the ineffectiveness of those remedies which we have been hitherto, and to a certain extent erroneously, taught to apply in all cases, early or late, the dilated and lax—paralysed—heart with overloaded veins, the cerebral anæmia, all point to this being a case of paralysis of the heart in a dilated and overfilled condition, but the amount of chloroform given—not more than twelve drops on the lint, which by no means represents the actual amount inhaled—is against the paralysis being due to the poison of chloroform. The empty state of the heart was probably due to the compression of the organ in our efforts at resuscitation.

MR. HORACE PECHELL read the following paper:

MR. PRESIDENT, LADIES, AND GENTLEMEN,—I propose this evening to offer a few remarks on ether anæsthesia, and to describe and demonstrate some methods of using Ormsby's inhaler, in which I claim (1) to provide a very simple method of rapid etherisation; (2) to combine the methods of the open and closed methods of administration, and to avoid the defects of both.

I also propose to read two brief notes—(1) on the posture of the patient for re-administration in a dental case, (2) and on the use of dilute solutions of chloroform in ether.

Ether anæsthesias may be classified as follows:

1. Those in which a larger quantity of ether is used, freely mixed with air, e.g. the American cone. Here, although there is little cyanosis, induction is long, and it is in other ways unsatisfactory. Still it emphasises a fact of prime importance, that ether is itself a pure anæsthetic—if given in sufficient amounts—without such adventitious aids as CO₂, N₂O, or A.C.E. mixture.

2. Those in which a very small amount of ether

is used, and air limitation is strictly practised, e.g. the Clover portable ether apparatus.

In this class cyanosis is regarded as a permissible if not a laudable condition, and the stage of induction is generally short.

3. Those methods in which the administration of A.C.E. or gas prefaces the exhibition of an overpowering dose of ether. The ether is here the agent used to complete and maintain anæsthesia.

Of this class there are three good examples :

1. A.C.E. followed by ether on the cone. This is an open method.

2. A.C.E. followed by ether in the Ormsby. This is a semi-open method.

3. Mr. Braine's plan of gas followed by ether in the Ormsby—an entirely closed method.

This classification is, you will observe, really a history of the progress of etherisation. Like the history of any other art it is one of excess, defect, and compromise.

This third period combines the heavy dose of the first period with the quick induction of the second.

The problem for the anæsthetist is, I believe, to add to this third class free aëration during induction without unduly prolonging the process.

In methods 1 and 2 the problem is solved by commencing the induction with A.C.E. mixture. Both methods work admirably in practice, but they both involve the use of a mixed anæsthesia, and, moreover, the successive use of two anæsthetics of widely different sections. They do, however, allow free aëration during the induction, are simple and easy to work, and fulfil every reasonable requirement for most purposes. For dental work they are both under the disadvantage of requiring the use of a chloroform-containing mixture, and dental administrations bulk so largely in a general practitioner's anæsthesias, that I think we must look further afield for a suitable method for dental surgery at any rate.

Of Mr. Braine's method I can only say that it demands so deep a knowledge of gas and of ether that I am sure it is not the method to put into the hands of a general practitioner unless he has had a large experience of all sorts of anæsthetics, and can appreciate the condition of his patient at any given moment. A method which, in a very short time, causes the patient to pass from the deepest

gas anæsthesia to the deepest ether anæsthesia, could not, I believe, be safely employed unless the anæsthetist were so able to judge of his patient's condition.

If we add to this the rigid air deprivation aimed at in this method, we have an additional complication. That all these difficulties are successfully overcome by a certain small class of very experienced administrators, all of us who have seen the method properly applied know very well.

When I describe my method you will see how much I owe to Mr. Braine's personal teaching, and that my method is a modification of his designed to avoid what I honestly believe to be its great fault, namely, that it does not provide for free aëration during the induction, and thus imposes a quite unnecessary strain on the heart.

Before proceeding to my own method I have two further topics to touch upon, namely, cyanosis and Ormsby's bag.

Cyanosis, oxygen deprivation, or more properly CO₂ poisoning, is not a good thing for a patient. Its ill effects may be completely disregarded in a single gas administration, but become (as Mr. Tyrrell points out) most serious in long operations. CO₂ is itself a protoplasmic poison. Its first effect on the respiratory centre is to irritate it, and later on to disorder, convulse, and finally paralyse it. Coincidentally with the irritation of the respiratory centre, increased blood-pressure sets in. This condition under ordinary circumstances is but transient, still it is not physiologically a satisfactory state of matters, and if it could be avoided so much the better. As I have just said, for a short operation the question of circulatory and respiratory derangement is more theoretical than practical, but for a long operation I believe it is a very practical objection, and that in such a case cyanosis, with its consequent heart strain, ought to be avoided from the start, even if at the cost of an extra minute or so in induction.

Ormsby's Inhaler.—The ideal inhaler should be cheap, simple in construction, light, easily worked by one hand, should supply any desired proportions of air and ether, and should have no narrow air-ways. It should be so made as to be easily taken apart for cleaning, and yet should when fitted together be practically in one piece. If a sponge is used, it ought to be impossible for it to freeze. Such an inhaler exists ready made in Mr.

Braine's pattern of Ormsby's apparatus. I have made a slight modification in it, which makes the air-way more free and allows it to be taken to pieces more easily. My friend Mr. Jones, of Alton, Hants, has got Messrs. Down Brothers to make him an Ormsby in which the bag and face-piece are connected by two bayonet-jointed brass collars. This is, I think, a useful improvement. Ormsby's inhaler has not enjoyed universal favour in the past; the sponge often froze, it wasted the ether, and more or less suffocated the patient if used by itself. In the method I am about to describe I believe I have got over these difficulties (and that without any essential addition whatever to Mr. Braine's very simple pattern), and that I have made the Ormsby act as a regulating ether inhaler.

The principle of my method is that an air current follows the path of least resistance. If I now take the inhaler without charging it and remove the valve cap, but leave the sponge *in situ*, and apply it lightly to the face and breathe, most of the air goes through the valve and the bag scarcely moves at all. On fitting the face-piece accurately, and on applying the valve and slowly closing it, the bag becomes more and more the way of least resistance, until when the valve is closed, to and fro breathing into and out of the bag is fully established. But as you will see by working the valve, the extent to which the bag is brought into play is accurately regulated.

To charge the Ormsby I remove the sponge and valve cap and hold the inhaler with the bag dependent. I then pour two ounces of ether into the bag and replace the sponge. I do not allow the sponge to touch the ether at all. Its sole function at first is to act as a porous diaphragm and divert the current of air from the bag. On applying the inhaler to the patient's face lightly most of his breath goes and comes through the valve, and he gets scarcely any ether at all. I now accurately fit the face-piece and replace the valve cap, so that its slits do not coincide and both admit air. The bag is now coming into action, and the patient gets more ether vapour. I then make the valve slits coincide, and if the breathing is quite free and unconstrained slowly close the valve. I then let the patient have eight or ten deep breaths to and from the bag, and then give a breath of fresh air through the valve, which I again close. I now manipulate the bag so that the ether is distributed over as wide

a surface as possible, and this, of course, increases the strength of the vapour. If all has been going well the inhalation up to this point will have taken about two minutes, and the patient will be breathing deeply and freely. I now gently tilt the bag so as to wet the sponge with a little ether. The sponge now for the first time becomes an ether distributor. If the patient tolerates this increase in the ether vapour, I presently hold up the bag and soak the sponge through and through with ether, and about a dozen deep breaths of concentrated ether will generally produce a good anæsthesia, lasting for ninety seconds or so. I average about three to three and a half minutes in induction. With a little care the patient will not hold his breath when the vapour is thus gradually strengthened. He may take one or two (not more) short breaths, but the rhythm and depth of the respirations are quickly resumed, and then the valve may be opened. I do not open the valve just when I am increasing the strength of the ether vapour, as I find the patient tends to cough if I do so.

I do not also wait for an entire loss of corneal reflex in an adult before allowing an operation to begin. If a patient can deeply and freely respire the concentrated ether given off from a well-soaked sponge in an Ormsby, he will tolerate anything. I believe Mr. Rowell was the first to draw attention to this as a sign of deep ether anæsthesia. I personally rely more on it (for dental anæsthesia) than on any other sign. I get very little if any change of colour, and by prolonging the induction a little and giving more air I can avoid it altogether.

I should like to point out that the preliminary passage of the breath through the sponge, warms it and prevents it from subsequently freezing. The moisture which the breath deposits on the sponge will raise the specific gravity of the ether, and consequently its boiling and freezing points. I have never known the sponge freeze since I took to charging the Ormsby as above described. I use a narrow-meshed abdominal sponge, rolled into a cone and projecting half an inch into the face-piece. This holds the ether well, and prevents it trickling on to the patient's face.

I believe the principle involved in this method is correct. It is the guiding principle of my class 3 of anæsthesias. It is that etherisation is in most cases best accomplished by administering the maximum of ether in the minimum of time

to a patient whose laryngeal reflexes are blunted by the preliminary exhibition of an adjuvant anæsthetic, be it A.C.E., gas, or dilute ether vapour. I believe the Ormsby is far ahead of any other bag in its power of supplying large volumes of concentrated ether vapour, and the capacious bag and wide air-channels of the Braine pattern are in themselves very effective preventives of cyanosis. The enormous dose of ether that can be got into a comparatively few breaths produces rapid anæsthesia, and allows of more fresh air than could be permitted in an apparatus such as Clover's, which cannot from its structure give so much ether per breath as the Ormsby. I therefore submit that my method supplies a simple plan of rapid etherisation—not a mixed anæsthesia—suitable for general practitioners, that it combines the free aëration of the cone with a rapidity and certainty of induction not inferior to that of most of the closed methods. I submit that it removes the only serious objection to Ormsby's bag, *i. e.* that it is not a regulating ether inhaler.

In giving gas and ether I employ a method which bears external resemblance to Mr. Braine's. I first give some gas, and then apply the Ormsby. In his method the patient passes from a state of deep gas anæsthesia to a state of deep ether anæsthesia. In my plan I make use of that property of gas which has given it the name of laughing gas. Supposing you take two or three breaths of gas, you do not notice much derangement of the sensorium until you have removed the face-piece and taken a breath or two of fresh air; then not only are the senses deranged, but the respiration is distinctly quicker and deeper.

That this happens after only a breath or so of gas, any one who has casually taken a mouthful will remember, but if one is put under gas for surgical purposes one does not feel any sensory or respiratory derangement until after many breaths.

In giving gas and ether I therefore give gas for about thirty seconds, and then apply the Ormsby—charged after my method—with both slits of the valve open, and close the valve as quickly as I can and push the ether. The ether vapour presented is so weak that the patient does not cough, and a breath or so of air so increases the depth and rapidity of his respiration and so deranges his perception that a rapid intake of ether vapour is insured. Once the valve is closed I proceed as

for ether only. I get good anæsthesia well under three minutes as a rule. I use gas, therefore, as an adjuvant anæsthetic and respiratory stimulant, not as an independent anæsthetic.

I will now pass on to my two brief notes on posture of patients in dental surgery, and on the uses of dilute solutions of chloroform.

I etherise my dental cases in the reclining posture. After the patient is unconscious and the dental surgeon has started work, I watch for signs of returning consciousness, and then immediately bend the patient's head forward into the bag to which I have added some fresh ether (on the sponge). I am sure the question of position of an etherised patient is too much governed by our chloroform prejudices. If it is right to sit up a blue and asphyxiated gas case, it cannot be wrong to sit up an etherised patient in a brief operation. When one bends the patient's head forward you put them in the instinctive (and physiological) posture of the drunkard, or of a man sleeping in the sitting position. This position is in daily use as a preventive of syncope.

Nasal respiration is assisted, the soft palate, tongue, lips, and cheeks fall forward, and fluids flow out of the mouth on to the sponge. As the fluids do not collect near the back of the tongue the patient does not try to swallow, and so will probably escape sickness during the anæsthesia, and will suffer less from it afterwards. There is also no time lost in sponging out the mouth, as by the time the patient is again ready for operation most of the clotted blood will be in the face-piece, and the bleeding from the gums will generally have stopped. Also as no time is lost the patient does not come round, and the laryngeal reflex is not recovered. Hence he is in a condition to respire concentrated ether, and another twelve to fifteen deep breaths will give you a full minute at least of perfect anæsthesia, with a quick recovery. Before reapplying the inhaler I put about two drachms of ether on the sponge. As I say, I adopt the sitting position if the person is healthy, but in a doubtful case I anæsthetise him in the lying-down position, and re-anæsthetise with the patient almost in the Sims' position.

Dilute chloroform.—The late Mr. Moss used to give A.C.E. on a Rendle's mask, after arranging a towel about the patient's face to prevent any air reaching him, which did not pass from the sponge.

He put four to six drachms on the sponge in one dose, and as soon as the patient respired freely pressed the mask firmly on the towel, and covered the perforations with the palm of his hand, and only allowed a little air to come in. This seems opposed to all teaching on the subject of the free ration of chloroform mixtures, yet he administered any thousands of times without a death.

For midwifery I use a mixture of chloroform in ether 1 to 7. I tie the Rendle's mask up in a towel, and put on the best part of an ounce. I do not think I ever have to wait more than three minutes, if as much, for complete muscular relaxation.

I also use the same preparation of chloroform and ether—from a Clover without the bag—after the first twenty minutes in a long operation. I got the idea from the practice at St. Thomas's, which in its final development has become the two-bottle method.

In conclusion, ladies and gentlemen, I beg to tender my thanks to the Council for their kind permission to read my paper, and to my proposer and seconder for the honour of membership. I do not apologise for choosing such a subject as ether administration, because I feel that with all its faults ether is our best and safest general anæsthetic. Anything, therefore, which will simplify its administration, and induce the general practitioner to give it a fair trial, will be of distinct service in reducing the anæsthetic death-rate. I thank you for the patient hearing you have given me, and must now leave my views to your kind criticism.

Mr. CARTER BRAINE said he agreed with most of the statements brought forward by Mr. Pechell regarding the Ormsby inhaler. He was a firm believer in that inhaler, and for ether anæsthetisation he possessed no other. With regard to the administration of nitrous oxide to deep anæsthesia, and then passing on to deep anæsthesia with ether by the Ormsby inhaler, he did not think Mr. Pechell's statement was quite correct, because there was an interval at the change of anæsthetics in which the patient held his breath, so that they could not exactly say that deep ether anæsthesia followed immediately after the nitrous oxide anæsthesia. It is true that patients become a little cyanosed at the change, but it will be found that there is sufficient air already in the bag of the

Ormsby to tide over that period between nitrous oxide anæsthesia and ether anæsthesia, and allow the patient sufficient oxygen for a minute or so, during which time he absorbs sufficient ether into his system to pass on smoothly to deep etherisation. It was his practice not to allow a whole breath of air in the interval; he gave gas to deep anæsthesia truly, but in applying the Ormsby he did so with the air-cap wide open, and gradually diminished the opening; this modification he had been trying for the last two years, and found there was very little if any cyanosis. With regard to the freezing of the sponge, that was always brought forward as one of the objections to the Ormsby inhaler. The freezing depended very much on the character of the sponge used, and also upon the quantity of ether which was poured on the sponge, and the manner in which the sponge was fixed in the cage. If they used a fine sponge, packed it tightly in the cage, poured a couple of ounces of ether on it, and the nitrous oxide anæsthetisation took one minute, then they would probably find that the sponge by that time was quite hard; but if an open sponge like a bath sponge were taken and packed loosely in the cage, and one ounce of ether poured on it, then freezing of the sponge would rarely occur. Formerly he used to warm the sponge before placing the ether on it, but during the last five years he had discarded this method, and used the sponge cold. Mr. Pechell advocated pouring ether into the bag—a couple of ounces. After hearing Mr. Pechell's account he had tried that method, and he must say it ruined his bag, and therefore he had not adopted the practice since. The idea of tilting the ether on to the sponge was a very good one, because theoretically they could give ether very gently and very quietly without cyanosis or struggling; but he, personally, would not teach this method to students, because if they happened to tilt a little too much ether on to the sponge, or if the sponge did not accurately fit the opening in the Ormsby, he would be afraid of fluid ether coming on to the patient's face, and perhaps even into the mouth, the consequences of which might be very serious. He considered the air opening in the valve of the inhaler passed round by Mr. Pechell was not anything like sufficient. With regard to the position of the patient in dental cases which had been advocated by Mr. Pechell, he thought they had all used that for a considerable

time in cases where several extractions had to be done, and the anæsthesia was not long enough, and so the inhaler had to be reapplied. The disadvantage of doing so was that the blood and mucus came on to the sponges and ruined them. Still it seemed to him to be the safest plan, and the easiest with Ormsby's inhaler. He formerly had used the Clover's inhaler in such cases, and upon tilting the patient forward found that blood was liable to run inside the dome, and in some cases completely occlude it.

Dr. SILK said he seldom used an Ormsby; he almost always employed a Clover. He would like to enter a very emphatic protest against Mr. Pechell's view that any form of etherisation ought to or was expected to produce lividity and cyanosis. He hoped and believed that all the members of this society taught and practised the contrary. When cyanosis was of any duration he himself believed that it was an indication of one of two things: either that the ether was being given badly, or else that the patient was not a fit subject for that particular drug. All the difference he could see between Mr. Pechell's method and that pursued by most administrators when using the Ormsby was that Mr. Pechell poured his ether into the bag and made very free use of the valve, whereas others poured the ether into the sponge, and instead of using the valves so freely, lifted up the edge of the face-piece, the object in either case being to aerate the ether vapour and not produce cyanosis. Mr. Pechell had also made a great point about carbonic acid poisoning. He quite agreed, as everybody must, that carbonic acid was in itself bad, but he thought they were rather apt to fly away with the idea that because a patient breathed in and out of the bag he must be breathing a large amount of carbonic acid. He did not take that view, because he thought it had been demonstrated that from the moment a patient breathed any anæsthetic vapour the tissue metabolism and therefore the formation of carbonic acid and other deleterious products was very much reduced. With regard to pouring the ether into the bag, he could only endorse the remarks of Mr. Carter Braine, that it was open to the objection of destroying the bag sooner or later, and that the drug was very apt to run upon the face of the patient, and he could not see any particular advantage in the proceeding.

With regard to the position of the patient, it had

been suggested that even when there was much blood and mucus in the mouth it was still possible to re-administer the ether, provided that the patient was tilted forwards, and this had been spoken of as a practice which they all knew of, even if they did not practise it. For himself he could say that he neither knew of it nor practised it. He considered that if the amount of mucus and blood in the patient's mouth was so great that there was fear that he might choke, re-administration ought not to be carried out. To be reduced to the necessity of pushing the patient forward seemed to him to be a doubtful proceeding. Under such circumstances he would much sooner wait until the following day. With the head well back, the small amount of mucus which he thought ought to be produced would run down the pharynx, and not hamper the operation in any way.

Mr. BELLAMY GARDNER said he would like to congratulate Mr. Pechell on the aim with which he had brought forward this modification of the apparatus for administering ether, because he thought anything which would make the administration of ether in this country more general than at present would be of immense advantage not only to the profession but to the public. Probably Mr. Pechell would make no claim that his method was perfect; its great objection was, no doubt, the large quantity of ether which he put into the bag, and which, in certain postures, would no doubt run back on to the patient's face. He had experience of this, because he had himself made a modification by which a little reservoir could be added to Ormsby's inhaler to regulate the amount of ether passing to the sponge. If the sponge was a little over-full the ether would run back into the face-piece. It was a matter requiring far more skill than Mr. Pechell had hinted at to know when the anæsthetic *had* run back. With regard to giving a *small* quantity of gas and then ether by this method, he thought Mr. Pechell had really hit the nail on the head, because undoubtedly in a great many cases in which he had tried the method of giving a *large* quantity of gas and then a full dose of ether there was a period in which the patient had not lost his laryngeal reflex; and if a strong vapour of ether were presented at the acme of a gas anæsthesia, and the patient then held his breath, an unnecessary strain was thrown upon the circulation. With regard to the administration for dental cases, he

disagreed with Mr. Pechell in putting the patient in a semi-recumbent position, because any mucus ran back immediately, and might cause slight hesitation in breathing, if not actual coughing. If the patient was upright or nearly so during the induction, and any difficulty arose, the hand of the administrator placed behind the head tilting it forward removed the obstruction at once. Moreover, if the tongue had slipped back, this proceeding brought it forwards. Where there were a large number of teeth to remove he thought the dentist would be disappointed if the whole matter were not finished at the sitting. To postpone the conclusion of the operation to the next day would mean probably considerable inconvenience also to the patient. In cases where the extraction of a large number of teeth was required, it was best to tilt the patient forward also when re-administering the ether. It was a routine practice in London for anæsthetists to give ether in the sitting posture without the slightest fear of syncope; though this was not the case with chloroform.

The PRESIDENT said he certainly agreed with Mr. Gardner that they must congratulate Mr. Pechell in his laudable aim to popularise the use of ether. If his methods did not altogether commend themselves to him, the speaker, it was probably because he was less familiar with them, and had not enjoyed the opportunities of perfecting himself in their use. He had used Ormsby's inhaler for a good many years, but he admitted he had never been able to satisfy himself as to its superiority over the method which was introduced by Mr. Clover. He could not help falling foul of Mr. Pechell in his historical references, because he assigned methods to various gentlemen who had only adopted them from their predecessors. It was surprising that anybody dealing with ether inhalers should have omitted to mention Mr. Clover as an exponent of a method of giving ether in the early days when chloroform was more in vogue than at present, and when ether was given seldom and badly. Most people knew of Clover's method, but very few knew it thoroughly, and in many cases Clover's principles as to its use were not strictly followed out. Personally, he would say, as Dr. Silk had remarked, that those who gave gas and ether following Mr. Clover's method, and achieved cyanosis, had much to learn, and had not done justice to Mr. Clover or his teaching. The

great point in favour of Clover's method, and it was a point which he understood Mr. Pechell had attempted to arrive at by the method which he had brought before them that night, was that the inhalation was commenced with a non-irritant body, nitrous oxide gas, then the nitrous oxide gas was allowed to pass over the ether and become slightly impregnated with it, but so slightly that although the anæsthesia was deepened no coughing was excited, and no cyanosis or spasm developed, and when the laryngeal reflexes were in abeyance ether was given in full strength without any fear of any of those untoward consequences which were commonly attributed to its use by those who were not familiar with Clover's method. It had already been said that the sitting posture for giving ether was one which was adopted every day and many times a day in London without any fear.

Mr. PECHELL, in reply, said he did not propose to introduce an apparatus which would supersede those used by specialists, but what he aimed at was to show them a method which he believed the general practitioner might be tempted to adopt.

He did not know whether specialists quite appreciated how very few general practitioners used ether at all. Clover's apparatus was costly, and was possessed by very few general practitioners, but Ormsby's apparatus could be produced very cheaply if there were sufficient demand for it. In the method described Ormsby's apparatus became quite as efficient a regulating inhaler as Clover's. Mr. Carter Braine had said that he administered with the valve wide open; this, however, was not the practice of Mr. F. W. Braine, who had personally taught Mr. Petchell to administer with the valve closed. It was also objected that the ether would rot the bag. The bag he exhibited had been in daily use for over a year, and showed no signs of rotting. Perhaps the fact that he always washed the bag out after use accounted for its good preservation.

With regard to the statement that ether would flow into the patient's mouth, this depended on the position of the patient and of the bag during the administration, and could not occur if the head were turned on one side and the bag were dependent. He had personally never known the ether to flow into the mouth.

When using Rendle's mask he tied it up in a towel so as to make a comfortably fitting face-piece,

and to restrict the air-supply. He had not suggested the position he advocated for dental surgery simply to avoid mucus collecting in the mouth; it prevented the free hæmorrhage which sometimes occurred from embarrassing the operator and the anæsthetist, and enabled the ether to be administered at the proper physiological moment without delay, and it avoided the necessity of sponging out the throat. When he spoke of dental surgery he did not mean the extraction of a single tooth, but rather the removal of a whole collection of stumps. In the country twenty or more stumps had often to be removed at a single sitting.

Mr. CARTER BRAINE, at the invitation of the President, exhibited an inhaler which he had been using for the last nine months. It was merely an Ormsby inhaler with various attachments which could be easily removed for giving either A.C.E. mixture, chloroform, or ether, throughout or in succession. It was all metal, beautifully clean, and all parts except the india rubber could be sterilised. The part which held the sponge was of wide calibre and incompressible, so that it did not become constricted with use. If the administrator found he was not getting on nicely with the A.C.E. mixture, and wanted to change to chloroform, he had only to remove the sponge and attach the tube of a Junker's inhaler to the A.C.E. attachment. He considered the inhaler he showed to be the nearest approach to an aseptic ether inhaler that they had. During the ten years he had been in practice he had worn out about six inhalers of the Ormsby pattern, but he considered that this pattern being practically indestructible would be of service to him to the end of his days. He would like to point out to Mr. Pechell his method of using this inhaler. He administered the nitrous oxide to deep anæsthesia, and then applied the Ormsby with the large valve open, so that the patient got air from the commencement of the ether administration; then the supply of air was gradually occluded until the patient was well off, so that in many respects Mr. Pechell and himself were working on the same lines.

REVIEW.

The Annual and Analytical Cyclopædia of Practical Medicine.—By Charles E. de M. Sajous, M.D., and One Hundred Associate Editors, assisted by Corresponding Editors, Collaborators, and Correspondents. Illustrated with chromo-lithographs, engravings, and maps. Volume I, 'Abdominal Injuries' to 'Bright's Disease.' Philadelphia, New York, Chicago: The F. A. Davis

Company, Publishers, 1898. Royal 8vo, pp. 611. Cloth, 5 dols.; half Russia, 6 dols.

The object of this useful publication is to furnish the medical public with a six-volume reference work, and thirty-six monthly supplements of Medical journalistic literature for a subscription of \$30. The volume to hand is excellently printed, and the text is not only beautifully illustrated by engravings, but chromo-lithographic plates and maps materially add to the value of this comprehensive and remarkable production. The subjects follow one another alphabetically, each being fully dealt with in a systematic and logical manner. Instead of presenting the excerpts from the year's literature arranged in order under a general head, each disease—including its sub-divisions: "Etiology," "Pathology," "Treatment," &c.—is described *in extenso*, and the new features that the year has brought forth are inserted in their respective places in the text. In this manner the reader is saved all fatiguing study; he has before him what before was left to his memory. The work, when completed, will present all the general diseases described in text-books on practical subjects—medicine, surgery, therapeutics, obstetrics, &c.—and, inserted in their logical order in the text, all the progressive features of value presented during the last decade. If the year brings forth nothing new upon any particular disease, the latter will, at least, appear as it was when last studied, whether this be one, two, five, or twenty years before. The general arrangement adopted will make it possible to cover the entire field in six volumes. The article on 'Abdominal Injuries,' for instance, contains 160 article excerpts besides the general text; that on 'Appendicitis' a still larger number. Being interpolated in the text and controversially arranged, the abstracts either sustain the views advanced or indicate fields as yet insufficiently explored. Confusion is avoided by using large type for the general text—that is to say, the description of a disease—and small type for the excerpts from journals. Either may thus be read separately. As to remedies, the list includes a few new agents which seem to merit further trial. Obsolete remedies have not been mentioned, the aim being to present those which constitute a modern physician's armamentarium. Again, only the diseases in which the remedies mentioned are of special value have been alluded to, along with what new points the recent literature may have afforded. The thanks of the profession are due to Dr. Sajous for his work, and want of space alone prevents us according his great undertaking a longer notice.

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HEPATITIS AND ITS TREATMENT,

A Clinical Lecture delivered at the Central London Sick Asylum to the Post-Graduate Class,

By JAMES CANTLIE, M.B., F.R.C.S.

LADIES AND GENTLEMEN,—The subject I have chosen for my lecture this afternoon is "Hepatitis and its Treatment." I should have preferred to have termed the lecture "The Treatment of Hepatitis" merely, but I must first touch upon those points which I consider essential in elucidating my plan of localising the inflammatory lesions in the region of the liver.

I divide inflammatory troubles in the hepatic area into (a) inflammation of the liver itself—true hepatitis; (b) inflammation of the capsule of the liver—perihepatitis; (c) inflammation between the layers of the broad ligament, a lesion to which I propose to give the name supra-hepatitis; (d) as a fourth class I would not forget the hepatic ducts, and the obstruction due to their inflammation.

I, however, propose to deal with the first three only, partly on account of the limited time at my disposal, and partly from the fact that biliary catarrh and obstruction chiefly affect the liver only secondarily.

I will not weary you with recapitulating the text-book signs and symptoms of hepatitis; these are well known to you all.

I. (a) Excluding traumatic causes, *hepatitis* is prone to occur in persons suffering from such (1) pathological conditions as are due to (i) obstruction, the result of pulmonary and cardiac venous engorgement; (ii) absorption of deleterious products derived from the abdominal organs; or (2) to physiological engorgement, the result of excess of food or drink. Primary hepatitis is well-nigh an unknown disease; and where no pathological conditions obtain, the preliminary state of physiological excess is well-nigh a constant quantity. Persistent use of excess of food will cause congestion of the liver; the addition of a quantity of alcohol

will induce an active hyperæmia; and it only requires a chill to set up inflammation. In tropical countries this is perhaps the commonest of all causes. With a minimum of exercise and a tendency to *bon vivant* which characterises most European residents in a tropical country, the liver is the organ which is most apt to suffer in consequence of chill. When the sun sets, the sudden cooling of the air causes a chill, and, to fall back upon a time-honoured expression, "the blood is driven from the skin to internal organs." In tropical countries one is apt to ascribe to malaria a number of ailments due to simple chill; and I am afraid we often traduce malaria by naming it as a cause, laying many things to its charge which are no doubt frequently induced by cold merely. The fact is we do not think of cold in hot countries. When we "catch" cold in temperate climates, the pulmonary organs are most frequently affected, but it is the liver that suffers in tropical regions, not the respiratory tract. It is to be remembered also that the temperature of the liver is higher than that of any other organ of the body, reaching 102° or 103° normally, and therefore most liable to chill.

I. (b) *Partial* or localised *hepatitis* almost invariably results from absorption of material by the portal system, from, say, the surface of an ulcer as in dysentery. Such a localised hepatitis is not unknown as a sequel of typhoid fever, though it is not a common occurrence. It may also result from malignant disease of rectum; ischio-rectal abscess; inflamed and ulcerated conditions of Peyer's patches, as met with in many tropical fevers; phlebitis of portal vein, &c., &c.

General and partial hepatitis compared.—Whatever the cause, *general* hepatitis causes the liver to increase in bulk and to become painful. The function of the liver being in abeyance, the bile is partly suppressed, and the venous engorgement of the liver and the consequent serous infiltration of the tissues being pronounced, partial obstruction to the minute biliary capillaries ensues. It will be seen that there is neither complete suppression nor complete obstruction in a general hepatitis, but the combination of the two serves to divert the bile from its usual channel and to cause a diminution of bile-pigment in the fæces, the presence of bile-colouring matter in the urine, and the appearance of a minor form of jaundice. The liver-cells are

working badly under the circumstances, and even the fluid they do produce cannot, on account of the swollen nature of the passages in the liver, get away. The small quantity of bile which is formed therefore is, as it were, thrown back on the blood, and attempts at elimination are tried by other organs; hence the bile-stained urine, the jaundiced conjunctivæ and skin, and the foul odour of the breath.

When the hepatitis is *partial*—that is, when localised points of inflammatory trouble exist—there need be, and, in fact, there generally is, no jaundice. So long as only a small part of the liver tissue is left fairly sound the bile is secreted and excreted with but little alteration. One half of the liver may be occupied by a huge abscess without there being marked symptoms of hepatic lesion, because the other half of the liver takes up the work, just as does one kidney when the other becomes diseased. Indeed, if one kidney is destroyed, the other will hypertrophy in obedience to the extra work required of it. In like manner, one half of the liver may so increase in weight, owing to the increased work thrown upon it, as to be found after death to be equal to the normal weight of the whole liver. In such a case, when disease attacks one half of the liver, one can find a marked difference in the size of the remaining half almost immediately, hypertrophy speedily showing.

In a paper which I read on the anatomy of the liver before the Anatomical Society at Dublin, in June, 1897, I showed that there was very little communication between the two sides of the liver; that when one injects the two main branches of portal vein, the two branches of the hepatic artery, and the hepatic duct with fluids of different colours, that there is no free (if any) communication between the vessels of the two sides. When no obstruction of the biliary passages is present, jaundice is not a marked sign of partial hepatitis; in fact, jaundice is so frequently absent that its presence or absence is one of the chief guides as to the extent of liver inflammation. Jaundice, slight it may be, is constant in general hepatitis; therefore its absence in any inflammatory disturbance of the liver marks the inflammation as partial only. We are very apt to assume that there can be no inflammatory hepatic lesion present because there is no jaundice. This, of course, is a great fallacy.

II. I will next take *perihepatitis*. That is a

condition which one frequently finds included under hepatitis, and treated with scanty consideration and extreme brevity. The frequency with which adhesions of the liver to neighbouring parts, more especially the diaphragm, are found in ordinary post-mortem examinations, would seem to indicate that it is a more common ailment than is generally allowed. Old-standing fibrous adhesions, admitting, by their length, of fairly free motion of the liver upon the abdominal walls and viscera, are met with in 30 per cent. of all post-mortem examinations in persons over thirty years of age. The pain so often ascribed to pleurisy pure and simple on the right side is, in many cases, caused in part by perihepatitis. Not that pain is a very marked symptom of perihepatitis at any time, for marked friction sounds may be heard in the subdiaphragmatic region, and yet but little pain is felt, except when a very deep breath is taken, or when the patient comes down smartly on the heels. This absence, or rather slight presence, of pain is no doubt the cause of perihepatic adhesions being found post mortem when no history of previous trouble in this region, or of pleurisy, accompanies it. In those cases diagnosed as pleurisy, in which no chest signs are found after death, we invariably find perihepatic inflammation, and, one ought to bear in mind the possibility of this fact, when treating low pleurisy on the right side. Perihepatitis from a chill is, if not a common, at least a fairly frequent condition. I do not feel inclined to set the disease aside as a secondary ailment, as is the case in most descriptions of perihepatitis. In right-sided pleurisy there will be found almost invariably adhesions between the liver and the diaphragm, but in the majority of cases this condition is neglected. Perihepatitis *per se* may occur from cold, just as does pleurisy. As with the liver so with the spleen; consider how often the spleen is adherent to the diaphragm—in quite 30 to 40 per cent. of post-mortem examinations. No doubt this condition is likewise due to a perisplenitis caused in the same way. We are inclined to pronounce the condition one of pleurisy, because friction exists, but one may also get friction from the peritoneum between the liver or spleen and the diaphragm, just as in the case of the pleura. There is a difference in the character of the rub in the two diseases. The rub of pleurisy is well-nigh con-

tinuous, whereas that caused by the liver rubbing against the diaphragm is intermittent, and is heard mostly at the end of inspiration or at the end of a deep expiration. A sufferer from pleurisy cannot take a deep inspiration, but with inflammation in the area of the liver he almost invariably can do so. Patients suffering from perihepatitis do not feel acutely ill; and they often persist in the statement that there is but little the matter with their liver—a very opposite condition to that we know of in pleurisy.

III. The next variety of inflammation to which I propose to refer is what I have ventured to call *supra-hepatic* inflammation and abscess—inflammation of the dome of the liver, in contradistinction to subdiaphragmatic inflammation and abscess. This condition I hold to be of primary importance in so-called abscess of the liver, but it is one which up to the present time has received no attention. The condition of the resulting abscess is usually classed as subdiaphragmatic; but I contend that the term is misleading, and founded on false premises. Writers, however, who deal with the matter, consider the disease to be the result of pulmonary trouble, and therefore secondary in its origin to disease of these organs. This is a conclusion I do not admit, but hold the opposite view, namely, that the inflammation beneath the diaphragm is the primary ailment (in the tropics, at least), and the lung trouble altogether secondary. The inflammation occurs between the layers of the broad ligament of the liver, and if suppuration results it is there that the abscess is met with. So pronouncedly is it kept within its limits, that in making a post-mortem on a case of so-called liver abscess, even where pus has been found during life, on first opening the abdomen and examining the liver one thinks that a mistake has been made, and turns to the thorax to find evidence of disease. Here, again, disappointment awaits one, for the costal pleura is seen to be healthy, and the visceral pleura presents a normal appearance. If the case is of long standing, however, it is found to be impossible to raise the lung from off the diaphragm, and one's attention is directed thither. On completing the examination the following condition is found: the broad ligament of the liver is seen to bulge, and on cutting into it pus or the seat of an abscess will be found between its layers. The liver substance is scooped out, but not deeply, to

form one part of the abscess wall; the diaphragm above, and the layers of the broad ligament in front and behind, complete the boundary. If the disease is of short duration, the lung will be found adherent to the diaphragm by ordinary pleuritic adhesions, but no pus is met with unless the abscess beneath has found its way through the diaphragm, and has allowed the pus to pass upwards through the lung and into a bronchus. Surely this is not to be termed a subdiaphragmatic abscess, and secondary to lung trouble. It is an abscess originating between the layers of the broad ligament, and involving the liver by an ulcerated surface. It is a supra-hepatic trouble pure and simple, and finds its way to pulmonary tissue only when, by pressure upwards, the pus traverses the diaphragm and lung tissue to reach a bronchus.

In this connection there is the ever-recurring discussion about the part played by dysentery. It is an every-day occurrence to meet with patients suffering from liver abscess who strenuously deny ever having had dysentery or even intestinal flux. Medical men as persistently attempt to prove a predysenteric state, and will not recognise that chill is a possible and the most frequent cause of the condition. The struggle to prove dysentery in every case of liver abscess goes on, although with but little success in many cases. The maxim that every case of liver abscess is secondary to dysentery is not true. I have seen a man, a new-comer from England to the tropics, develop a supra-hepatic abscess within fourteen days of his arrival in Hong Kong, who never visited the tropics before,—in fact, had never been out of England, and who never had dysentery. No doubt abscesses in the liver *substance* are mostly preceded by dysentery, but there is a form of abscess in connection with the liver which is totally independent of dysenteric trouble. It has its origin at the top of the liver, between the layers of the broad ligament, and may be termed abscess of the dome of the liver, or supra-hepatic abscess. It is the abscess of the liver which occurs independently of intestinal troubles, and its non-recognition has caused all the controversy on this subject.

It will help to elucidate the pathology of this condition to consider how jaundice occurs. Supposing the bile-ducts of a dog are tied, jaundice will supervene. First of all there are clay-coloured stools, then saffron-coloured urine, then jaundice of

the skin and mucous membrane, and foul breath. The bile is trying to escape by the excretory organs; but how does it find its way into the blood? One is apt to think that it is sucked into the branches of the hepatic vein; but it is doubtful if that is the path by which it gets thither.

By experiment it has been proved that if the thoracic duct of a dog is tied after the common bile duct has been ligatured there will be no jaundice; but if the thoracic duct is not tied jaundice will ensue. Therefore the bile finds its way out of the liver into the thoracic duct, and the thoracic duct pours it into the blood. But if the thoracic duct is kept tied for a while the jaundice will reappear (V. Harley). This is owing to the fact that the left thoracic duct is not the only channel by which the contents of the duct are carried. There is the right lymphatic duct as an open channel, which will in time develop and help to transmit the bile. If, however, the right lymphatic duct and the thoracic duct are simultaneously ligatured no jaundice will appear. How do the lymphatics of the upper part of the liver leave the organ? They do so through the space between the layers of the broad ligament all round the inferior vena cava. Here, then, may be the explanation of supra-hepatic abscess, namely, a lymphangitis of the supra-hepatic vessels.

The symptoms of supra-hepatic inflammation are distinct, and easy to diagnose when once the condition is admitted. It starts suddenly with pain in the region of the lower chest wall, resembling pleurisy. The temperature rises with or without a rigor to 102° or more in a few hours; there is distress in breathing, a slight catching cough, and a quick, rather hard pulse. The patient professes not to feel ill, and a sort of "stitch" on deep breathing—not the excruciating pain of pleurisy—is all that he complains of. There need be no sickness, no appearance of bile in the urine or skin, nor is bile absent from the fæces. From the onset there will be found characteristic local signs, evident on percussion. On following out the upper border of the liver, it will be found that in the region of the nipple line there is a sharply rising conical area, dull to percussion and destitute of breath-sounds. The base blends with the liver dulness below, but the apex and sides are surrounded by lung tissue, which is resonant at first, but at an early date the patch becomes congested,

and gives an impaired note on percussion which varies from day to day, or almost from hour to hour, with the varying amount of congestion. The liver is not affected except by being advanced forwards and downwards, but it is not painful on light palpation or percussion. When, however, the liver is grasped and moved by placing one hand over the region of the twelfth rib behind, and the other hand in front of the abdomen, rather severe deep-seated pain will be elicited. At the same time a never-failing symptom is complained of during the movement consequent on examination, namely, a shooting pain within the acromio-clavicular angle at the top of the right shoulder. Some people say that shoulder pain is worthless in diagnosis, but in most liver inflammations this can be elicited by movement, and what is held to be a doubtful converted into a positive symptom. Moreover, after one has examined the liver as indicated, the patient will complain of pain in that one spot for some time after the examination, and will press the thumb into the hollow, seeking relief thereby. When the inflammation between the broad ligament layers has gone on to the formation of pus, there may be but slight distress, the patient being unwilling to believe that any such serious trouble as a "liver abscess" can be present.

I have endeavoured to show that there are three distinct primary seats of inflammatory conditions in the region of the liver—hepatic, perihepatic, and supra-hepatic; that any one state may induce a partial involvement of the other, but that every one may exist primarily as a distinct lesion, differing in cause, in symptoms, in signs, and in course from its allies.

Treatment.—The first essential to procure in all inflammatory conditions in the region of the liver is rest. A man suffering from hepatitis due to alcohol is a restless soul; he says he does not feel ill enough to lie down, and he does not credit the statement that he is seriously ill. Even when pus has formed the patient feels and looks fairly well, and it is very difficult to persuade him to take rest. The object of insisting upon rest is to prevent the diaphragm moving over the top of the liver, and it is necessary to place the patient into the horizontal position, so as to take the weight of the arms off the chest, and thereby allow thoracic breathing to proceed more freely. In hepatic lesions of an inflammatory nature the patient uses the upper

part of the chest solely, so as to avoid moving the lower part of the thorax and the diaphragm. Hepatic inflammatory troubles do not of themselves cause very much pain; it is the compression of the liver between the descending and advancing diaphragm that starts the greatest amount of pain. If this can be allayed, patients with hepatic, supra-hepatic, or perihepatic ailments have no great suffering. To fix the lower part of the chest and thereby procure the fullest amount of rest and freedom from pain, it has been proposed to apply strapping to the side of the chest over the liver. This is a very excellent means of allaying movement, and thereby procuring freedom from pain, and at the same time lessening the to and fro movement. By applying hot fomentations or poultices over the liver there is no doubt that alleviation of local inflammation is obtained. But the poultice must be a huge one, covering the side of the chest from the spine to the middle line in front, and in depth it ought to reach from below the axilla to the brim of the pelvis. This should be changed every three hours. It is of little use painting the right side with iodine. Local applications of nitro-hydrochloric acid are favourite remedies for liver troubles; baths of nitro-hydrochloric acid are also advocated, but I am not of opinion that they possess a specific virtue. Should an acid bath be decided upon make it in this way: Take half a pint of the dilute nitro-hydrochloric acid and dilute it with one gallon of water at a temperature of 100°.

Food.—If mechanical rest is advisable in cases of liver disease of an inflammatory nature, how much more is it essential to procure a diet that will allow of physiological rest. One of the functions of the liver is to aid in the digestion of food; secretion being its work, it is evidently as unfair to demand the organ to secrete as it is to expect an inflamed knee-joint to bear the weight of the body. What food will allow of the liver attaining the maximum of rest? It is the fashion—I cannot call it by another name—to put the patient on milk, let the ailment be what it may. Now milk is not a natural food for adults at any time, and to call upon the liver to digest milk, more especially when it is inflamed, is a mistake. Were the liver trained to accommodate itself to deal with a huge quantity of milk from childhood upwards, the case might be different; but no adults of the

human race use milk as a diet, nor even as the principal part of their diet. In fact, it is only the children of the peoples of Northern Europe that are given milk after their babyhood. Milk is by all adults considered to be "bilious," yet it is the custom in even hepatic trouble, when the liver has struck work, to administer this "bilious" food. The only form in which a milk diet is admissible is in the form of whey. Starchy foods ought also to be withheld from the dietary. During digestion they flood the liver with material which adds to its difficulties in righting itself. Animal food in its most digestive form is the key-note of successful dieting in hepatic inflammatory derangements. Enough ought only to be given to support life so long as acute symptoms remain. Raw meat juice, raw meat itself, chicken soup, and weak beef tea should form the elements of diet. As drinks, hot water, and weak freshly-made tea with lemon, are the best. As the acute symptoms subside the "meat" nourishment may be increased, and the first starchy food to be administered ought to be well-steamed rice.

Medicinal treatment.—It is customary and wise to clear out the bowels by a purge. For this purpose calomel is the best drug to use, as calomel is supposed to relieve the liver by causing a flow of bile. Let us consider what this really means. Here is an organ that has struck work, and the function of secreting bile is, like other functions, in abeyance. It therefore does not seem sound therapeutics to drive the organ to do work. But calomel does not cause the liver to *secrete* bile; it merely stimulates the surface of the duodenum, and causes the gall-bladder to empty itself by reflex action. The copious bilious stool brought down by calomel is merely the bile which has been stored up in the bile passages. Moreover, calomel has an antiphlogistic action upon all inflammatory tissues, which gives it a claim to be administered in hepatic inflammations. Three grains of calomel given on an empty stomach is perhaps the best form of initial purgative in hepatitis. If the bowels are not freely moved after twelve hours have passed, then, but not as a matter of course, give a saline draught. For this purpose sulphate of soda is the best, and sodii sulphas effervescens is the best form, half an ounce in a small quantity of water. The quantity of water should be small if we desire to procure

the full physiological and therapeutic action of the drug. Some advise giving nitro-muriatic acid to make the liver act, but this is not sound treatment in the acute stage. Acids in the active stages of the disease can only do harm by engorging the liver. The best way of abating the congestion is a neutral salt of some kind. Neither should alkalies be given, for they cause an increased flow of gastric juice; the sulphate of soda mentioned only drains the passage. Chloride of ammonium in twenty-grain doses, either by itself or in combination with the iodide and bromide of potassium, may be made into a mixture in conjunction with taraxacum. Experience really is the main indication for these drugs being given, for experimental therapeutics have failed to prove how they act. When pus is suspected, or has actually formed, what is to be done? The answer is very precise—aspirate, tap, and drain. If, indeed, one has to treat a case of simple hepatitis that proves obstinate, again would I recommend aspiration. Percuss out the liver, find the seat of irregular and abnormal dulness, and then aspirate. Do not hesitate to insert the needle, the danger to the lung or diaphragm is a mere "bogie." In the first place the lung is usually pushed up well out of the way, but even if the lung is punctured in all probability good will result. No harm has ever come to a patient with liver inflammation from this treatment. Insert the needle to a depth of four or five inches. If by the first puncture no pus is found, the operation may be repeated again and again. Suppose no pus is found, does good result from the procedure? Undoubtedly so, and I believe the explanation of this is, that from the liver punctures blood escapes freely into the abdominal cavity. This leads to no untoward circumstance, as the following case shows:—Whilst tapping a man for ascites, with the idea for facilitating relief to his enlarged liver, I inserted an aspirating needle into his liver and drew off a little blood: the ascitic fluid meanwhile flowing through the cannula, introduced below the umbilicus. At first the fluid was clear, but when I withdrew the aspirating needle from his liver the fluid became deeply tinged with blood, and in this state it continued until the whole of the abdominal cavity was drained. The patient made a rapid improvement; and although it is beside the question, I may remark in passing that I tapped this man in

all seven times, with the result that the patient completely recovered.

If on aspiration one strikes pus, withdraw the needle immediately. The aspirator is used only as a means of diagnosis, not of cure. Moreover, if you reduce the size of the abscess much, the more difficult will it be to strike the abscess by the trocar, the use of which is the next step. Along the track followed by the needle insert the trocar and cannula I now show you. This looks a huge weapon, but its use is attended with no danger. The instrument can penetrate to a depth of six inches, the measurements being indicated by circles one inch apart on the metal. The point being triangular, pushes aside rather than cuts the structures penetrated. Now withdraw the trocar, leaving the cannula in the side. Pus will flow from the cannula, but it is wise to prevent it flowing away until the next step is taken. Through the cannula insert this piece of india-rubber tubing. Use a large tube—as large as possible; one, in fact, much larger than the cannula. But how is the wide tubing to be introduced along the narrower cannula? To do that I employ a method first shown me by Dr. Manson, but I use a much simpler plan of introducing the tube than he does. Over the 10-inch long steel rod I show you, with a projecting hook at the one end, stretch a piece of tubing nine inches long until it is sufficiently reduced in thickness to pass along the cannula. When the bottom of the track is touched slip the cannula out, passing readily over the stretched tube and the enclosed rod. Let go the stretched tube, withdraw the rod, and you will find that the tube expands, and completely filling the wound, allows the pus to escape along its channel. The pus should be conducted by a long tube to a basin of water filled with some weak carbolised solution. It is a good plan to introduce a glass (bent) tube some four or five inches long of the same calibre as the tubing, so that the flow of pus may be watched and noted as to rate, colour, consistency, &c.

Before closing my lecture I wish to bring to your notice a very handy and a very accurate method of recording the size and position of abdominal organs or tumours. I have named it the "Keith method," as the plan was first shown to me by Mr. Arthur Keith, M.B., F.R.C.S., of the London Hospital Medical School. It con-

sists in first carefully percussing out the organ, say the liver, for that is the organ I have used this plan mostly for. I mark the limits of percussion dulness on the skin by black paint (Indian ink). Then with red paint I map out the ribs by a broad band drawn on the skin over each rib. When the colouring fluids have dried, I place a piece of thin, transparent muslin over the front of the body, large enough to cover the body from the clavicles to the pubes. With red paint I trace on the muslin the red lines over the ribs, indicate the arch of the subcostal angle, the nipples, the umbilicus with the same, and finally draw the brush along the black outlines of the liver as they are seen through the muslin. On the muslin paint the patient's name and the date of drawing, as well as the disease, for future reference and comparison with note-book. By this method one preserves a "life-sized" drawing of the liver, and a permanent record. The drawing from its accuracy is of immense advantage to a patient going home from the East, or even coming up from the country for treatment, as the doctor he consults can, by laying the map on the patient's skin, perceive at once what change, if any, has taken place. We are, of course, familiar with the diagrams traced on paper, but they are almost microscopic in dimensions, and by them the size of an organ or tumour cannot be accurately gauged. I would earnestly recommend for your acceptance the "Keith method of recording abdominal conditions."

Therapeutic Uses of Gastric Juice.—Dr. Fremont ('Gazette médicale de Paris,' May 7th) communicated to the Congress of Medicine at Montpellier the results of some experiments on the therapeutic uses of gastric juice taken from the stomachs of dogs. He records a case of acute enteritis cured in a few hours, one of cholera nostras cured in twelve hours, and among others related are *grippe* with gastro-intestinal complications, gastro-intestinal dyspepsia and enlargement of the liver with progressive emaciation, *apepsia* in a morphino-maniac with gummata and exostoses, deficient assimilation in a three-months-old child, typhoid fever with severe emaciation, and dilatation of the stomach with progressive loss of flesh. These cases were all cured by this means.—*N. Y. Med. Journ.*, June 4th, 1898.

WITH MR. SYMONDS IN THE OUT-PATIENT DEPARTMENT OF GUY'S HOSPITAL,

May 12th, 1898.

GENTLEMEN,—Here is a man aged 23 with a mass of glands on the right side of his neck. You will notice that they are multiple, movable, elastic, and that they are not fixed to the surrounding connective tissue. They are under the sternomastoid, and you can feel their softness and mobility. Now what may such a condition be due to? First, it may be either primary lymphatic glandular trouble, or it may be secondary disease. It is easier to exclude the secondary disease first. If it were secondary, it might be secondary to some disease in the palate, or pharynx, or the nose, or the mouth; and the trouble to which it may be secondary may be carcinoma or sarcoma, simple irritation, and tubercle. Tubercle may be either primary or secondary. Now what primary disease might it be? It might be sarcoma, lympho-sarcoma, or lymphadenoma. Which is this? There are no enlarged glands on the opposite side of the neck. The fact that the mass is large and the glands multiple and movable and painless are points against tubercle; so also is the fact that the enlargement is on one side of the neck only, and the absence of evidence of the tubercular process about the mass. Then, tubercular glands degenerate early; the life of tubercle is a short life. Tubercular glands caseate early, and get fixed to the surrounding structures. Now what is against lympho-sarcoma? If it were sarcoma it would by this time be infiltrating, and more adherent than it is; also there would probably be some sign of cachexia in the patient, but there is not. Now we come to lymphadenoma. It is like lymphadenoma because of its mobility, its elasticity, its softness, and the fact that it is limited to one side. Therefore we may describe the case as one of lymphadenoma of the neck. Now what is the progress? The progress of tubercle is generally towards caseation and suppuration; and the progress of lympho-sarcoma when it is as big as in the case before you is towards ulceration, involvement of the skin and adjacent organs, and

there are signs of pressure and suppuration in the superficial parts. The progress of lymphadenoma is towards enlargement and increased pressure only. If it goes badly it extends up and down the neck and involves the other side, and is associated with general adenia. Another point is that it may get better. This patient, you hear, thinks he is decidedly better; and I should agree with that, and say that he has improved very much indeed under treatment during the last month. What should be given for treatment in such cases? Arsenic, cod-liver oil, bone marrow, and perhaps iron. Every day this patient has a fresh marrow-bone; it is very lightly cooked, and is then spread on toast and butter. Therefore I take it that the result of treatment supports the diagnosis. This case is exactly similar to one which has been under my care for some time, and is gradually improving. I do not think iodide does much good in these cases; this form of glandular enlargement gets well with arsenic. We shall press the arsenic in this man. [On May 26th the glands were much smaller; reduced a good half in the fortnight.]

The next patient is a man aged 28, with a left varicocele. We know it is a varicocele because it is a swelling on the left side of the scrotum above and around the testes. It is composed of thick, coiled, individual veins. Notice that you can empty the tumour on pressure; on pressing it in this way with the whole hand it empties slowly, quietly, and noiselessly. If you take it in your hand and ask the patient to cough you will get the impulse, and this impulse is due to the venous pressure. If such a patient lies down the swelling is very much diminished, because the blood goes away and the thickened veins alone remain. Now you might possibly mistake such a condition for omental hernia. What kind of omental hernia can it be taken for? The irreducible omental hernia—a piece of omentum which represents the end of the great omentum, and is adherent to the bottom of the sac and unaltered in character; generally in young people of from fifteen to sixteen years of age. When omentum remains a long time in a sac it becomes thickened and ultimately fibrous. Why? The omentum becomes thickened because it is compressed by the neck of the sac. You know that the neck of a hernial sac tends to close spontaneously, and this closing of the neck upon the

omentum leads to congestion, and this in turn leads to exudation of serum and cells; then follows organisation and fibrous changes in the omentum. If the omentum is sufficiently compressed it may become as hard as a carcinoma, for which I have seen it mistaken. Therefore when you are giving a diagnosis between varicocele and hernia you must specify what kind of hernia you mean, and at what age it generally occurs. This man's occupation is that of a waterside labourer, and the condition has been present some years. He says it aches when he is lifting a heavy weight, and, of course, his occupation consists in lifting. These facts are sufficient to justify operation. What should the operation be? The varicocele should be freely excised. Operators vary as to the length of the veins they remove. I usually operate through an incision three quarters of an inch long, isolate the mass, draw out the veins, ligature them high up, and tie again about one inch above the testicle, and then cut out the intervening portion. The ligature (of catgut) should be left long at each end, and the two pieces tied together, so as to bring the two severed ends into contact. This draws up the testis, and we have a better result. That is a suggestion of Mr. Bennett's. Astley Cooper's test for varicocele is to reduce it while in a lying position, to put the finger over the external ring, and get him to stand up. If it is varicocele it returns; if it is hernia it will not return. Of course you do not want that test unless it is an omental hernia.

The next case is that of a boy aged 13, who comes with a history of having fallen upon his shoulder, and the subsequent development of a lump in his axilla. That is the information with which the patient comes in. The lump, you see, is at the upper end and inner side of his humerus; it is hard, connected with bone, and fixed. It is slightly smaller as you approach the bone, indicating that there is a kind of neck or isthmus. Another point which you will notice is that the free surface is lobulated and lumpy on the surface. That description is sufficient to enable you to make a diagnosis. It is an exostosis of the upper end of the humerus. What is the lobulated portion composed of? It is cartilaginous, and the neck is bone. An exostosis does not grow at the free surface, but at the junction of the bone and the cartilage, where there is an irregular ossifying layer.

The increase takes place by proliferation of the cartilage and ossification between the junction of the bone and of the cartilage. That fact tells us how we can cure these cases. It is sufficient to cut through the neck, for by doing this you take away the growing line; but it is better to chisel it off the surface of the bone. I must contrast that with an exostosis of the big toe. In the latter the exostosis is composed chiefly of connective tissue, besides bone, in which there is very little or no microscopical cartilage. You must cut out a piece of bone, or else the growth will come back again. This form, because of the recurrence, has been called sarcoma. I have cut up an entire exostosis and examined perhaps sixty sections; in only three or four was there hyaline cartilage.

When you are asked about the treatment of exostosis you must bear the points I have mentioned in mind, and contrast that with those which resemble the exostosis of the big toe, and for which you must cut away a piece of bone, and with the ordinary one, such as in this patient's arm. In the museum you will find great toes which have been amputated for exostosis, the growth has been looked upon as a recurrent sarcoma, and in which the recurrence has been due to imperfect removal on the first occasion. The exostoses illustrated by this case occur in young people at the epiphyseal line, and in order of frequency they occur in the lower end of the femur, the upper and inner end of the tibia, and in the upper end of the humerus; that is to say, the humerus is the third commonest seat of these exostoses.

The next patient is a man aged 62, who has a malignant stricture of his œsophagus, thirteen inches from the teeth. When he first came he was able to swallow, and I could not get anything through the stricture. A little later he became unable to swallow anything at all; but I was able to insert this long feeding-tube—a No. 8—through the stricture. Through the tube we have been able to feed him. After this tube was in for a week, it so much dilated the stricture that the patient was able to swallow by the side of the tube, taking such things as custard, beef tea and other fluids, cauliflower and other substances (in this way). This has been going on for five weeks, and during that time he certainly has not lost flesh, which is an important point, and he is stronger. The tube he is wearing is a gum-elastic. To-day

he comes complaining that he has a little sore throat and cough, and thinks he has caught a little cold. But the more correct reading of these symptoms is that the tube is probably irritating the mucous membrane over his cricoid, and probably causing a little ulceration in this position, and giving rise to the symptoms of cold and irritation. That is one objection to these long tubes, which were first introduced by Krishaber, of Paris, and here by Mr. Croft and Mr. Durham. It was this inconvenience which led me to suggest the short tube, but I have not been able to put in a short tube in this man. But now that the stricture has been so much dilated, I shall try to put in a short tube, and I hope he may be able to dispense with the longer one. It is very desirable to remove the irritation to his cricoid. So far he has no secondary symptoms. The commonest secondary symptoms of malignant stricture are pulmonary gangrene, perforation into the trachea, and food pneumonia, or perforation of the aorta. I have seen two instances of the latter. You could not have failed to notice the hoarse, watery sound of this man's voice when he spoke to us; and that is due to the swelling of the aryæno-epiglottic fold. Therefore beware of accepting a patient's statement that he has a cold when he is suffering from malignant stricture.

[On May 19th a No. 10 four-inch Symonds funnel-tube was inserted, and when seen on May 26th the man was comfortable and swallowing well.]

The next patient is a man aged 31. You see he comes to us with a very much swollen knee, with an opening of the nature of a sinus on the inner side of the knee, and one on the outer side. The edges of the apertures are undermined and thin, the base having a grey gelatinous appearance like a connective-tissue slough. He says his knee has been bad for three years, but he has managed to walk upon the leg until three weeks ago. The knee broke—or in other words, the abscess discharged—ten days ago. You see that the hollows of his knee are filled up, and the material which makes the part large is hard. Yet it is to a certain extent elastic, especially at the sides. There is no fluctuation in it. The material composing the enlargement in a joint may be due to bony deposit, thickening of the synovial membrane itself, or to excessive secretion by that membrane, or again

there may be disease of the connective tissue around. You can satisfy yourself, by feeling it, that there is no fluid.

Now what are the causes which make the synovial membrane thick and hard? Practically they are two—tubercle and syphilis; and a part affected with these may be large also because of fluid being in the joint, but here there is no evidence of fluid. There may be granuloma in the synovial membrane, and that is due to tubercle and syphilis as far as we know, and to nothing else.

Is it bone then? If you are trying to make out bony enlargement, examine the lower third of the femur; if there is any thickening of bone it will be found there. When you have the articular ends, as in this case, covered with thickened synovial material, you cannot say whether the bone is thick or not. Therefore the only way you can make out thickening is to examine the bone at some distance from the synovial membrane, where it is uncovered. If you find this part of the bone is normal in size, you can safely say that the bone is normal at the knee-joint. Though that statement is not quite orthodox, I think you may take it as true. That there is no enlargement can be proved by making sections through such a joint as in excision. Probably you would find this man's knee would be half an inch larger transversely than the other knee, but it would be found to be entirely due to the synovial membrane.

I have noticed that we never get thickening of the articular ends externally—that is to say, by external additions—unless there is chronic suppuration, and then you do get osteophytic deposits and thickenings, that is, by additions to the surface. If you have disease in the centre of a bone, as, for instance, a tubercular sequestrum in the lower end of the femur, a tubercular sequestrum big enough to occupy the entire end, you will have no enlargement of the bone externally; it is said that in articular osteitis the bone is "expanded," but I do not find this statement borne out by observation, for if you cut up the bone and make a transverse section you will see that it is not enlarged at all. In this patient I do not think it would be found that the bone is enlarged, therefore we will exclude that; so we are practically left with thickening of the synovial membrane as a cause of his trouble. This granuloma of the knee with infiltration of the

synovial membrane is due either to tuberculosis or syphilis. Which is it in this case? There are two sinuses which discharge thin pus. Even with this amount of disease and enlargement he is able to permit very slight movement, and is able to walk. Would he be able to walk if it were tuberculous? Yes; and the condition taken with the presence of the sinuses is in favour of tubercle. That it is possible for the patient to walk in tubercular arthritis is shown by the fact that the tubercular process in the knee is in the subendothelial layer, and that the tubercular material undergoing caseation may work outwards, and may not go inwards towards the joint. A probe passed through this sinus may enter the subendothelial layer without penetrating the joint. So long as the tubercular process keeps away from the interior of the joint, there may be but little pain, and you may have a dozen sinuses round the joint without one of them entering its cavity, and so long as that condition exists there may be little pain. The moment the process invades the inside of the joint you have pain and fixation. If early in the course of the disease the process enters the joint, you get tubercular synovitis and all the pain which attends disease of the joint. This man probably has localised tubercle of the synovial membrane, at least in two points, and there is little doubt that the inflammation has passed outwards through the skin, so that there is little disease in the articulation itself. All the surrounding oedema may be consequent upon disease of the capsule itself. Always remember that, even in a case of tubercle, there may be a syphilitic basis. One of the great characteristics of syphilitic joint is its painlessness, and you may remove the local disease and the patient get quite well. Since last week this patient thinks he has got better, but I do not like the look of it myself.

What is the prognosis? How far has the disease advanced? You will see that the leg is slightly rotated outwards, and I think it is also a little displaced backwards, which indicates that the ligaments of the joint are becoming softened from oedema, and so outward rotation is taking place from the action of the external rotators. The tubercle will probably soon enter the articulation, and then there will be a good deal of pain and suppuration. The best advice to give the patient is to recommend excision, which would give him a

good leg, though a stiff one. The sinuses make the forecast less favorable, but they are not against operation. There is no history of rash, and nothing about the patient to suggest syphilitic disease.

This young man came to us with a history of having jumped from a cart a week ago, when he experienced a sudden pain in the calf. He walked about on it as long as he could, and finally came here with a calf swollen and very tender, the tenderness being chiefly on the inner side of the centre of the calf, where we found a lump. The first diagnosis given was that he had phlebitis, and the second that he had a ruptured gastrocnemius. The evidence of the latter was localisation of the swelling and tenderness, and local thickening, showing that there was extravasation of blood. The line of treatment adopted was to keep the patient at rest, the part being bandaged up with lead lotion, and he is now, at the end of a week, practically well. What was his danger if he had continued to walk about? The chief danger was that he might have an abscess; from that I think he has just been saved by treatment and proper care. The injured muscle is now healing, and I think all danger of further trouble from it is passed.

This little girl is an interesting case. She was in a hospital with scarlet fever. She got laryngitis and ulceration of her throat, which were so bad that she had to be tracheotomised, and she has never been able to dispense with the tube. Dr. Goodall sent her here to me. I divided the thyroid and cricoid cartilages, and found the larynx so filled with fibrous tissue that it was practically obliterated; the destructive ulceration from the scarlet fever and the subsequent cicatrization had practically obliterated the lumen. I made an attempt to put one tube upwards into the larynx, and another downwards into the trachea, so as to make the child speak, but it was not very successful. She cannot speak at all. I propose to make another attempt to put in a kind of artificial larynx,—that is to say, a downward tube into the trachea and an upward tube into the larynx. The difficulty of putting a tube upwards is owing to the cricoid, for after extirpation of the larynx you have no cricoid, and you put the tube up through the soft structures, but here it is such a long way back before you can

turn upwards that I found adaptation impossible. The question is, whether it would be wise to remove the anterior part of the cricoid so as to make a more easy track. Remember it is possible to phonate with the lateral walls of the pharynx. It is the first case I have come across in which the tracheotomy tube could not be got out after it has been worn for some time.

This has been a very unfortunate patient; during her illness she had gangrene of the tips of the fingers; she has lost half of the forefinger of the right hand, the tips of two other fingers and the little finger, and since the attack of scarlet fever her ears have emitted an offensive discharge. You will see there is some ulceration of the skin around the opening for the tracheotomy tube.

Velander's Pillow-slip Method of Mercurial Inunction.—Dr. R. Hogner describes this new procedure of the Stockholm professor, Dr. E. Velander, for the application of mercurial ointment. He considers it to be practical, effective, convenient, and clean. Velander was convinced some years ago that the mercury in an ointment is absorbed after being vaporised. He has, therefore, given up spreading the ointment upon the skin, and spreads it instead upon the inside of a small pillow-case, which is hung upon succeeding days alternately on the back and on the chest. It is kept in position by ribbons over the shoulders and around the waist. The pillow-case is made of cheap cotton cloth, and one of the external faces is smeared with the ointment. It is then inverted so as to bring the ointment on its inner surface, and is placed in position so that the layer of cloth upon which the ointment is spread is in direct contact with the body, while its unsmeared side protects the clothing. Every night fresh ointment is spread over the old without removing this, and at the end of ten days the pillow-case is thrown away and a new one is prepared. Baths should be taken twice a week, and the skin of the chest and back washed every night. The quantity of ointment used daily is 6 grams (90 grains). The effect of the treatment is very rapid, as more mercury can in this manner be absorbed with fewer unpleasant effects, such as stomatitis, &c., than when it is otherwise administered.—*Boston Medical and Surgical Journal*, March 31st.

A CASE OF ACUTE INVERSION OF THE UTERUS.*

BY

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British Medical Association.

CASES of acute partial inversion at labour becoming complete on the fourth day are of sufficient rarity to encourage me to bring this case to the notice and kindly criticism of our branch.

The case occurred in a young woman aged 24, whose occupation before marriage had been that of a domestic servant. She had been four years married, and had had four children. The first was stillborn at full time, the second premature, and the others are still alive.

She had always been exceptionally healthy, had menstruated regularly, and had hardly ever had backache.

She was confined of her third child a little more than twelve months ago, was attended by a midwife, lost much blood at the time, but made a good recovery. On January 20th, 1898, she was confined by a midwife of her fourth child. Second stage was completed after seven hours' labour, and after waiting an hour, the nurse, finding the placenta not forthcoming, is stated to have passed her hand right inside and pulled it out. This caused much pain and a severe loss of blood, patient becoming greatly exhausted. Contrary to the rule in her previous confinements, she had much bearing down pain and continual cramps in her legs. Three days after her confinement she got out of bed on to the commode, but failing to pass anything at first, she strained, and felt something like a child's head coming down, causing her great pain. She was assisted into bed, and gradually, with a good deal of bleeding, the mass passed outside the vulva. I saw her in conjunction with Dr. Thompson the same day, January 23rd. She was well nourished, but very pale, breathing hurriedly, with a very weak pulse and much collapse. On examination a mass about the size of a foetal head was

* Read before the West Somerset Branch of the British Medical Association at Taunton on March 25th, 1898.

seen protruding from the vulva. It seemed as if it were attached to a broad pedicle about as thick as my wrist. The surface of the mass was ragged and bleeding, and bits of adherent placenta were visible upon it. The patient being so exhausted, we considered it best not to administer chloroform; and after drawing off the urine, we took it in turns to compress the inverted fundus and try to return it.

This was no easy task, though the vulva and vagina were very roomy; and after manipulating for twenty minutes the fundus was reduced into the vagina. Pressure with one hand in the vagina was continued, the patient being in the genu-pectoral position, and the mass gradually became smaller until the fingers could be felt between the os and the inverted fundus. Finding after half an hour's kneading that no further progress was being made, we decided to send her to the infirmary (three miles off). She bore the journey well. Temperature on admission was 102° F.; pulse 120. The vagina was douched with iodine tinc. 3ij to 0j, and a temporary pad of sal alembroth gauze inserted, together with an ordinary cup-and-sterm pessary to prevent further inversion.

January 24th.—Patient had a fair night. Temp. 100° , pulse 94. Vagina douched every four hours. Vulva beginning to be sore. I inserted an Aveling's repositor, which I had wired for.

January 25th.—The repositor was found next morning to have slipped, so it was replaced, but again that evening had slipped past the fundus. To prevent this a pad of lint was placed between it and the fundus. Patient now taking nourishment well.

January 26th.—To-day the cup again slipped, evidently being too small to receive the fundus. It was therefore removed, and under the supervision of Dr. Barford, the house surgeon, who was unremitting in his attention, a suitable cup two inches and a half in diameter was made and fitted on to the repositor. Discharge less foul. Enema given, which acted fairly.

January 27th.—The larger cup was introduced, and answered well. Temp. 100° , pulse 98.

January 28th.—Uterus now found to be gradually receding, discharge much less, temp. 99° , and urine passed voluntarily. The larger cup was removed at night and the smaller one reintroduced. Uterus could be felt above the pubes for the first time.

On January 29th, seven days after admission, the inversion of the uterus was completely restored, repositor being found inside os, but able to be withdrawn easily. General condition of patient improving. Temp. 98.8° .

January 30th.—Cervix contracting up, admitting only two fingers. From this time the uterus diminished in size. Vagina douched twice daily. Still slight sanious discharge.

On February 5th, seventeen days after admission, twenty from confinement, the temperature rose to 101° , with tenderness over pubes.

On February 10th temperature fell to 99.4° , and it has remained below that level since. A large ring pessary was inserted, and the patient left the infirmary on March 5th, forty-five days after admission.

Remarks.—When first seen the mass protruding from the vulva looked somewhat like a fibroid polypus, but the history, bimanual examination, and the presence of placental tufts, proved its real character. Manipulation also caused much pain, and the patient's collapse was extreme. The small cup of the repositor first used merely caused a cupping of the fundus, and failed to reduce it, but the larger cup seemed to steadily reduce the fundus at its cervical attachment. There seems no doubt that a partial inversion occurred when the placenta was extracted, and that a further descent, ending in complete inversion, occurred when at stool on the fourth day.

Water in the Treatment of Gastro-intestinal Disorders.—Lightly shows by experiments upon himself and others that when ten to a dozen tumblerfuls of water are taken in twenty-four hours, there is a decided and constant effect upon the activity of the eliminating organs. In cases of chronic gastritis, lavage, or drinking of water has the happiest effect. Where there is much dilatation of the stomach water may be given in small doses often repeated, also in hyperchlorhydria.

In chronic constipation the condition is frequently better relieved by the ingestion of large draughts of water than by any other means. In the treatment of intestinal auto-intoxication water, taken in large quantities, is of value, especially in causing increased elimination.

Medical News, April 16th, 1898.

NORTH-WEST LONDON CLINICAL SOCIETY.

Ordinary Meeting held at the North-West London Hospital,

Dr. CUBITT LUCEY in the Chair.

Primary Spastic Paralysis.

DR. JAMES TAYLOR showed a man æt. 27, who came to the out-patient department of the National Hospital, Queen Square, when he was found to be suffering from weakness of both legs, more marked in the right. The history was that the condition had existed for eighteen months, and had gradually become worse. When examined he was found to have exaggeration of the knee-jerks on both sides and ankle-clonus on the right. On inquiring into the family history, he (Dr. Taylor) ascertained that the patient had a sister who was affected in a similar way, whom he had since seen. She was rather worse than the present patient, and had suffered for some years from increasing weakness in the legs. She also had exaggerated knee-jerks and ankle-clonus on both sides. The case was clearly one of spastic paralysis, and from the fact that another member of the family was affected in the same way without the presence of any bone disease or any history of acute myelitis, it seemed clear that the condition was one of a family type of spastic paralysis. Besides this evidence of affection of the lateral columns in the present case he also had evidence of a developmental defect elsewhere in the existence of a coloboma of the iris and choroid in the left eye. That was the first instance of the kind he had seen, and, as far as he knew, it was the first case which had been described in which, in addition to the evidence of mal-development in the spinal cord, there was also evidence of mal-development elsewhere. On that account he thought the case might be interesting to members of the Society. It was some years since family spastic paralysis was described, and it formed a condition analogous to the condition of Friedreich's ataxy, in which there was defect, more particularly in the posterior columns of the cord. It was quite possible that both the family type of lateral sclerosis, and what one might speak of as a family type of posterior sclerosis (Friedreich's

ataxy) were essentially the same kind of disease, depending in the one case on the involvement preponderatingly of the lateral columns, and in the other of the posterior columns. If it had not already been done, it was possible that some one might find cases forming a connecting link between the two. Some years ago he published in the 'Practitioner' the description of a case which in every essential point resembled the cases of Friedreich's ataxy in the presence of ataxy and lateral curvature of the spine, but differed from the ordinary cases of Friedreich's disease in having the knee-jerks present. In Friedreich's ataxy the sclerosis seemed to fall with special emphasis on the posterior columns, and in such a condition as he referred to they might imagine a general sclerosis such as occurred in Friedreich's ataxy, but with the emphasis falling on the lateral columns.

Dr. Taylor said he also brought with him, in the hope of illustrating what he had said about the case, an instance of Friedreich's ataxy. The subject was a girl of fifteen, who began to have some difficulty in walking at five years of age. When she was aged eleven she was said to have had St. Vitus' dance, but he was inclined not to attach much importance to the history of that, because often in the out-patient room cases of Friedreich's ataxy were mistaken for chorea on account of the tremors and movements which were present. In the present patient, although the movements were present, they were not at all the movements of chorea. She also had characteristic deformity in the foot, namely, turning up of the toes and a high arch of the foot. Her knee-jerks were absent. In her family there was no other instance of the same condition. She had very marked lateral curvature of the spine. There was practically no defect in the upper extremities. The sensation was perfectly good, and there was no affection of the sphincters, nor yet in the first case.

Dr. GUTHRIE thought the cases were particularly interesting, especially the one which illustrated the family type of spasticity. Sometimes one met with cases in which the spasticity was congenital, and not developed towards the end of the first decade as usual. Two or three years ago he showed before the Society specimens of this type of disease in a family of five children, three of whom were more or less similarly affected. After some trouble

he found that the father was the subject of an identical condition. The paralysis affected the *tibiales antici* on both sides, and there was spasticity in the arms and legs, and exaggerated knee-jerks. He agreed with Dr. Taylor's explanation of the condition.

Paralysis of Left Leg.

Dr. HARRY CAMPBELL showed two young women the subjects of paralysis of the left leg, the paralysis in each case being due to pressure. The first was a woman of twenty-three, who had been suffering from pain in the left leg for two or three years. There was foot-drop on the left side, and considerable paralysis of the muscles supplied by the sciatic and anterior crural. These muscles exhibited the reaction of degeneration. There were areas of anæsthesia and intense pain. Until recently the knee-jerk on the left side, in spite of the paralysis of the quadriceps, was present, and indeed exaggerated, but it was now disappearing. As the affection was confined to one limb, it was obviously not a pure toxic paralysis; some local cause must be at work.

The second patient also had paralysis of the left leg, but the muscles had not been tested electrically. She also had a bad pain shooting down the leg, and had areas of anæsthesia, but not so marked as in the other case. This patient kept her leg semi-flexed, but the other did not. Mr. Durham had satisfied himself that there was no disease of the hip-joint, and none could be discovered in the pelvis. In the first case about six weeks ago a swelling appeared over the left buttock, which Mr. Durham thought was an abscess in connection with the sacro-iliac joint, but on cutting into the swelling it proved to be a sarcoma. No tumour had been discovered in the second case, but there must be something pressing upon the sacral plexus. He thought the cases showed the curious susceptibility of the nerve-fibres supplying the extensors of the foot; these muscles being paralysed more than any others, as happened in toxic paralysis. It was proposed to try the effect of Coley's fluid on the first case; and in regard to the second he would be glad to hear the opinions of members as to the cause of the condition.

Dr. LEONARD PARRY suggested as a possible explanation of the involvement of the anterior

crural a secondary deposit in the glands, even though involvement of glands was not common in sarcoma.

Dr. JAMES TAYLOR thought it possible that the distance of the fibres causing extension of the foot from the central cell might explain their peculiar susceptibility to pressure just as they were to toxins.

Idioglossia with Pseudo-hypertrophic Paralysis.

Dr. GUTHRIE showed a boy *æt.* 7, a very typical case of pseudo-hypertrophic paralysis. He brought the case forward to illustrate the peculiarities of speech. Several cases of so-called idioglossia had been described. What was really at fault, in his opinion, was the hearing.

Dr. JAMES TAYLOR thought the association of idioglossia with pseudo-hypertrophic paralysis was extremely interesting. He did not think they were connected in any way. But it was a wonder that more of us did not speak as that boy did, because of the gibberish which nurses and mothers talked to their children. The talk of the present patient strongly suggested that of a nurse when speaking to children. He had the privilege of seeing one of Dr. Hadden's cases at St. Thomas's Hospital some years ago. He was very much worse than the boy before them, although he was older, and the improvement after two months' tuition, which the sister gave him, was most remarkable.

Dr. LEONARD PARRY said he had to thank Dr. Guthrie for bringing the case forward, as it enabled him (Dr. Parry) to make a diagnosis in a case he had recently seen. It was a boy of five, whose speech was affected in just the same way. His brother, when a few years old, spoke in the same manner; but now, at twelve years, his articulation was quite normal. There was no pseudo-hypertrophic paralysis in his case, and he would be interested to know whether the association of the two affections was common.

Dr. SUTHERLAND said he was interested in Dr. Guthrie's explanation, which was more feasible than that which the mothers put forward, namely, that the child was tongue-tied. Still Dr. Guthrie's explanation did not seem to him to quite meet the circumstances, because children suffering from idioglossia presented such similar defects of speech

that the condition could not be due to defective hearing.

Dr. GUTHRIE, in reply, contended that the peculiarities of speech were not exactly the same in every case, and that defective hearing did not necessarily imply deafness.

Pseudo-hypertrophic Paralysis.

Dr. GUTHRIE next showed (for Dr. Turner) a case of pseudo-hypertrophic paralysis. The patient was a young woman æt. 20, a dressmaker by occupation, and she had had some difficulty in walking and in getting up and down stairs for eight to ten months, and the condition had been getting worse. She had no difficulty of micturition and no pain in the legs, but was easily tired, and complained of a pain in the lower part of the back. The gait was not typical of any one disease. The muscles reacted well to faradism. There was some hardness about the right calf, and the knee-jerks could not be obtained. The plantar reflex was present. There was a fine tremor of the arm, but no intentional tremor. There was slight nystagmus on external deviation of the eyes; the muscles were flabby. She had considerable difficulty in raising herself from the sitting posture, and if told to sit on the floor she could not resume the standing posture without assistance, and then had to lever herself up by placing the hands on the knees. There was considerable weakness of the extensors. There was no apparent hypertrophy, yet he had no doubt it was the same disease as in the other case.

Intestinal Obstruction in an Infant, due to Rectal Spasm.

Dr. LEONARD PARRY said he had an infant under his care presenting a condition of great rarity. He had been unable to find records of any similar case, or to find a consultant of experience who had seen one like it. The only point of interest in the family history was that after her third confinement the mother was attacked by puerperal insanity, from which she recovered, but had one or two relapses, which were connected with some pelvic disturbance. This was the fourth child born in a normal manner; but from the first day of its birth it was seized with attacks, accompanied by the following symptoms: its abdomen began to swell

considerably, the child was in considerable pain, vomited frequently, and there was absolute constipation. On examination nothing abnormal was discovered in the abdomen except the uniform swelling. The vomiting was not fecal—simply a bile-stained fluid. The finger passed easily up the rectum for one and a half to two inches, and then seemed to come on a termination of the rectum. On moving the finger about and exercising gentle pressure something seemed to give way, and a large quantity of liquid dark-stained feces flew out a distance of six to eight feet, as if propelled by a syringe. After that the abdomen subsided, the pain passed away, and the vomiting ceased. But the condition recurred at regular intervals for the first four months, and the only way to get a motion passed was by introducing the finger into the rectum and exercising gentle pressure. The most common cause of intestinal obstruction in an infant was intussusception, some congenital defect in the development of the lower bowel, or, rarely, strangulated hernia. There was no malformation of the rectum, and he could only attribute it to spasm. Every now and then the child also suffered from attacks of retention of urine, evidently due to some muscular spasm of the urethra. He had been of the opinion that the child would grow out of the condition; it was now six months old, and appeared to be doing so, as it could sometimes pass a motion in the normal manner. The child had been treated with good doses of recognised antispasmodics, such as bromides and conium, but they had no effect upon it. Circumcision was recommended and adopted, but practically no benefit ensued.

Mr. JACKSON CLARKE suggested that as there was also some retention of urine, there might be a pouch at the termination of one or other ureters. In dissections of children who had lived a certain number of years he had seen such pouches, of sizes varying from that of a Tangerine orange to an ordinary orange. If such a pouch were there, filled periodically with urine, it might, by pressure on the rectum, cause occlusion. In the duodenum there was sometimes a large diaphragm which was perforated by a relatively small orifice, but it was difficult to account for the presence of a similar structure in the rectum, though he thought a mechanical cause rather than spasm probably gave rise to the condition present in Dr. Parry's patient,

which was one of rare interest, and he hoped the future course of the case would be watched.

Dr. SUTHERLAND thought it would be rather unusual for a spasmodic condition to persist so long and continuously as apparently this rectal stricture had done, and he asked if there were no occasions on which the spasm seemed to subside. He thought it would be interesting also to know whether the constriction were a regular annular constriction. Although antispasmodics had had no effect, he suggested that Dr. Parry might try rectal injections of chloral, which would overcome most conditions of spasm, and were well tolerated by infants.

Dr. E. CLAUDE TAYLOR suggested that the cause of the obstruction was mechanical, probably caused by matter collecting behind projecting abnormal intestinal valves.

Dr. PARRY briefly replied, stating that in the earlier months there were no motions passed in the normal way. He pointed out that the constriction would not be annular if it were caused by pressure of a tumour from the outside. Moreover, no mechanical cause would leave the walls normal afterwards. He still believed it was pure muscular spasm, probably in a neurotic child.

"Ingrowing" Toe-nail.

Mr. JACKSON CLARKE, in the course of a discussion on the above subject, said the term "ingrowing" toe-nail was unfortunate; it should be "in-pressed" toe-nail, or the equivalent of that. Speaking from a fairly extensive personal experience, he did not hesitate to say that this condition did not require any operation at all. With proper boots and antiseptic dressings he had never failed to obtain a cure. Some of the patients he had treated had had avulsion of the nail performed as many as ten or twelve times before coming to him, and the new nail, though deformed, was still *in-pressed* by the badly-shaped boots. The indications were to attend to the boots and to simple matters of cleanliness. No operative treatment was called for.

We have received from the firm of H. K. Lewis a copy of the ninth edition of Martindale and Westcott's 'Extra Pharmacopœia.' The principal changes in the work are due to its revision to bring it into accordance with the recent official Pharmacopœia.

NOTES, ETC.

Etiology of Cancer.—Roswell Park gives a *resumé* of this question, in which especial reference is paid to the parasitic theory. He quotes largely from the papers of Sanfelice and Roncali, and states that they have discovered a fungus belonging to yeasts as the active agent. This germ belongs to the Blastomycetæ. They reproduce themselves by budding, and have the form of young cacti. It may be stated positively, he says, (1) that some of these germs may be isolated by culture methods from certain carcinomata and sarcomata; (2) that they belong among the yeasts; (3) they will produce tumours in animals by injections under certain precautions; (4) from these tumours further cultures can be made with which inoculation can be practised.

He also states that Gussenbauer has been able to obtain from seven cases of melano-sarcoma an anaërobic coccus, which grows in ordinary culture media.—*American Journal of the Medical Sciences*, May, 1898.

The Treatment of Insomnia in Children.

Comby ('La Med. Mod.', 1897, viii, 249.)—Soporifics are not indicated in infants when insomnia is due to improper food or to disturbances of digestion. In such cases we must remove the cause. Insomnia is often met with in children who are given alcoholic potions, coffee, tea, &c. In these cases a cure is obtained by withholding the cause. Some children sleep restlessly on account of eating too much nitrogenous food; these children should be allowed meat only once a day. If the cause is not found in feeding, insomnia may be due to nervous causes. Before having recourse to soporifics, physical anodynes should be applied; for example, warm baths before bedtime of fifteen to twenty minutes' duration. In some children cool baths, or even douches, will exert a more favourable influence. Finally, we may use the wet pack advantageously two or three times a day in cases of marked cerebral irritation. In children, whose brain is very active, rest to the latter should, of course, be prescribed. Should all these measures prove insufficient, it may become necessary to administer

soporifics. One of the simplest of these remedies is orange flower water, which may be prescribed in quantities of twenty to sixty grammes (5 to 15 drams) before bedtime. This remedy frequently induces quiet sleep, lasting the whole night. Opium should be administered, but only in small doses, when insomnia is due to cough or pain. The bromides are always indicated when neurosis, accompanied by cerebral irritation, is present. Bromide of potassium or sodium may be given in doses of 0.10 (1½ grains) in sweetened water, or in syrup or milk. If the child is unable to swallow these remedies, they may be administered in clysmata or in suppositories.

Chloral hydrate in doses of 0.05 (¾ grain) in the first year of life is safe and active; in larger doses it has a bad action on the heart. It may be used in solution, in enema, or in the form of suppositories.

A combination of bromide of potassium and chloral is quite effective:

℞ Potassii bromat.,
 Chloral hydrat., āā ... 2.0 (½ dram)
 Extr. hyoscyami ...
 Extr. belladonnæ,
 Extr. cannabis indicæ, āā 0.02 (¼ minim)
 Syr. flor. aurant. ... 30.0 (1 ounce)
 Aq. destill. ... 40.0 (1½ ounce)

Sig.: A coffeespoonful every hour.

The disulfones employed in recent years are indicated in children. Sulphonal may be given internally or in clysmata in doses of 0.10 to 0.15, to 0.25 (1½ to 4 grains) at a dose, according to the age of the child. If sleep is not induced after one or two hours the dose may be repeated. As regards trional, Claus recommends it in children from one month to one year old in doses of 0.20 to 0.40 (3 to 6 grains), in children between one and two years of age in doses of 0.40 to 0.80 (6 to 12 grains), in those between two and six years in doses of 0.80 to 1.20 (12 to 18 grains), in children between six and ten years of age in doses of 1.20 to 1.50 (18 to 23 grains). Comby considers these doses too large, as he has seen the temperature fall from 38.5° C. to 34.0° C. in a greatly excited girl ten years of age, suffering from meningitis, after the exhibition during the day of 1 gramme (15 grains) of trional, divided in four doses.

According to his numerous experiments with

trional, this remedy proved to be an excellent hypnotic in doses of 0.25 to 0.75 (3¼ to 11½ grains), which was well borne and showed no bad after-effects in these quantities. This dose may be repeated every evening, but it would be better to omit it every other night, for the reason that sleep is frequently permanently induced after one or two doses of trional.—*Pediatrics*, June, 1898.

The Place of Formation of the Cholera Antibodies.—R. Pfeiffer and Marx accept Ehrlich's ingenious hypothesis of the formation of antitoxins, which, it will be remembered, is that the antitoxins are formed by the same organs which have a specific affinity for the molecules of the toxins. The antitoxin, according to Ehrlich, is nothing else than that cell-constituent which combines with the toxin. Through the combination of the toxin molecule with the "toxophorous" lateral chains of the cell protoplasm a deficiency is produced which acts as a stimulus, and leads to the excessive formation of the used-up substance, whereupon the surplus of this substance enters the blood and acts as an antitoxin. The experiments of Wassermann lend distinct support to this theory. But Pfeiffer and Marx do not think that the theory explains the formation of specific antibodies, *i. e.* bactericidal bodies. There is no reason to think that the cholera antibodies exist preformed in the normal organism.

Their experiments were designed to reveal the place of formation of these substances, the starting point of their researches being the fact that a single injection of a dead culture of cholera vibrios into strong young rabbits is followed, in the course of a few days, by a marked specific blood change.

They first investigated the question as to whether the leucocytes were the bearers of the bactericidal substance or not. Without detailing their interesting experiments, suffice it to say that neither the leucocytes of the blood nor those of inflammatory exudates are the carriers of the protective bodies.

They then studied the activity of the juices of various organs, and found that the spleen, bone-marrow, lymph glands, and possibly the lungs, contained larger quantities of active substances than were demonstrable in the circulating blood. The spleen, indeed, had active power before the blood showed any bactericidal action. Removal

of the spleen did not interfere with the prompt development of bactericidal bodies, probably, the authors think, because other organs (marrow and lymph glands) unfolded a vicarious activity.—*Zeitschrift für Hygiene und Infektionskrankheiten*, Band xxvii, Heft 2, April, 1898.

Dressing for Fractured Clavicle.—Henson recently presented before the Richmond Academy of Medicine an improved method of treating fracture of the clavicle. He says that the strip, in the Sayre method, which passes from the sound shoulder underneath the opposite elbow and back again, does its work well when first applied, but is almost certain to slip, and in warm weather very quickly; is apt to roll up on the shoulder and become cord-like, and is altogether very uncomfortable. In place of this strip he has, out of denim, unbleached jeans or other very stout cloth, a shoulder-cap fashioned for the sound shoulder. On this he has two extensions or tails, one opposite the other. This must be made to fit the whole shoulder and the upper part of the chest just below the axilla snugly, and so an arm-hole is necessary, which also serves to prevent slipping. There should be almost twice as much of the cap upon the shoulder as below the axilla. The posterior extension directed across the back along a line drawn from the shoulder supporting the cap obliquely toward the opposite elbow, should reach a little beyond the median line, and may be pointed. The anterior tail has the same course across the front of the chest, reaching as far as the opposite nipple; its end should be from one and one half to two and one half inches in width, according to the size of the patient, and upon its under surface a pocket is made, reaching from near its tip upward and outward quite to the top of the shoulder, and just wide enough to accommodate the patient's hand. With tension made upon both extensions at the same time, in the line of their direction, the cap should bear equal pressure upon the shoulder and chest. To each extension he attaches a buckle. Another strip of the same kind of cloth is taken,—narrow, oblong, sufficiently wide to accommodate the elbow, and long enough to reach from the middle of the arm to the middle of the forearm, when extended,—and to each end of this a tape is attached. As to application, first the horizontal adhesive strip is applied, just as in the Sayre

method, then the shoulder-cap is fitted upon the sound shoulder. Then into the pocket of the front extension introduce the hand of the injured limb, after carrying the forearm up across the front of the chest. Buttons may be used in place of the buckles.

Richmond Journal of Practice, April, 1898.

The Cautery in the Removal of the Appendix.—Dr. A. J. C. Skene publishes in the *New York Medical Journal* of March 5th, 1898, an article on the use of hæmostatic forceps susceptible of being heated by the electric current to a degree sufficient to cauterise in cases in which the appendix has to be excised. His method is at least suggestive. We quote his description of an operation.

“For the first time in the history of appendicectomy the method of operating with the electric hæmostatic forceps was used. This departure from the current methods of ligature, suture, cauterisation, invagination, and others is the logical outcome of the success of Dr. Skene's practice when operating upon the pelvic viscera. All the other steps of the operation were such as are advised by surgeons generally. The incision was the ordinary one over McBurney's point, two inches in length. On inspection, both the appendix and the meso-appendix were found to be much enlarged and thickened, and superficially traversed by numerous dilated blood-vessels. There were no adhesions. The first grasp of the forceps was upon the meso-appendix, close to its mesenteric attachment. A current which heated the forceps to 180° F. was then induced for half a minute. Upon the removal of the forceps the tissues were found to be not charred, but dried, having the appearance of white horny matter. Scissors were used to bisect this desiccated area. A second seizure was made upon the appendix itself close to the caput coli, and the same current continued for ninety seconds. The forceps was then removed, and the tissue divided in the line of the desiccated area away from the caput. The same result was manifested. No charred tissue, no bleeding, and more important than all, no escape of the contents of the appendix. The tissues had simply been dried out. Just at this point a rather violent attack of retching came upon the patient, which continued for nearly a minute, yet without inducing any change whatever in the

stump. All the severe pressure and strain had not forced even a speck of blood or serum into the compressed area. The abdominal cavity was left perfectly free from any foreign matter whatever. Sutures and dressings as usual. Time of operation, fifteen minutes."—*Medicine*, June, 1898.

Captol: a New Antiseborrhoeic.—Eichhoff in the 'Deutsche med. Woch.' recommends captol, a condensation product of tannin and chloral, in seborrhoea capitis and its complications, such as itching, scale formation, and thinning of the hair. Captol has more rapid and definite effects than a mixture of the two drugs from which it is made, and is entirely harmless and unirritating. It forms a dark brown hygroscopic powder which dissolves with difficulty in cold water, but more easily in a mixture of warm water and alcohol, is not affected by acids, but is decomposed by alkalis and turns dark. Eichhoff employs it in a 1 to 2 per cent. alcoholic solution, and asserts that in every case in which he has employed it his results have been wonderfully good. In eight to fourteen days the scales have disappeared from the scalp, the hypersecretion of the sebaceous glands has ceased, and the falling out of the hair has stopped. He employs the preparation called "spiritus captoli compositus," which has the following formula:

Captol,
Chloral hydrate,
Acid. tartaric., 33 1.0 gram.
Ol. ricini 0.5 "
Spirit. vin. (65 per cent.)... 100.0 "
Essent. flor. æth. 9.5 "
Journ. Amer. Med. Assoc., May 28th, 1898.

Thallium Acetate in Night Sweats of Phthisis.—Combemale, at a recent meeting of the Academy of Medicine, spoke highly of acetate of thallium in the night sweats of phthisis. He has had only one failure in the treatment of thirty patients. The remedy also exercises a beneficial effect upon chronic catarrh, due to bronchial dilatation, emphysema, &c. The daily dose is one to two grains, given in pills, about one hour before bed-time. It should never be given during more than four successive days.

Medical News, April 2nd, 1898.

Modification of Schede's Thoracoplasty in Cases of Empyema.—Sudeck ('Deutsche Zeitschrift für Chirurgie,' Bd. xlvii, Hefte 2 and 3, 1898) proposes what he considers a serviceable modification of Schede's operation of thoracoplasty. Schede's operation he describes as follows:—An incision is to be made beginning at the outer border of the pectoralis major at the level of the fourth rib, curved downward to the lower margin of the pleural cavity, then upward, outward, and backward to the middle of the scapula, which is thrust out of the way by turning the arm upward over the breast. This flap is raised and the whole chest wall, including the second rib, is resected. The remaining cavity is filled in as far as possible by a musculo-cutaneous flap. The greatest difficulty is experienced in so arranging this that the apex of the pleural space is carried in. There often results a persistent fistula.

This operation is usually performed on extremely weak tuberculous individuals. It is therefore important that the operation should be as simple as possible, and it would seem advisable to complete it at several sittings.

Sudeck's method is as follows: The first incision begins at the cartilaginous insertion of the fourth rib, and runs horizontally outward and backward across the scapula as far as the vertebral arch. Parallel with this incision a second is made on a level with the floor of the suppurating cavity. These two incisions are connected in the axillary line by a vertical cut making the figure H. The two flaps on the bar of the H are then turned forward and backward. The ribs are resected from the tenth to the third inclusive, and the thoracic walls, together with the thickened pleura, are removed throughout the whole extent of the cavity as far as the second rib. In spite of the shrinkage which occurs in the flaps, when they are spread out it will be found possible to nearly cover the remaining exposed pleural surface, the tongue-like flaps being stretched past each other. They are held in place by packing, and the part which is uncovered remains an open surface. At the second sitting this can be covered either by transplantation or skin grafting. At the third operation the depression made by the costal pleural at the apex of the lung can be closed either by transplanting the flaps or by resection of the second rib.—*Therapeutic Gazette*.

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A CLINICAL LECTURE

ON

FIVE CASES OF TABES DORSALIS.

Delivered at Charing Cross Hospital, March 3rd, 1898,

BY

FREDERICK W. MOTT, M.D., F.R.C.P., F.R.S.,

Physician in charge of Out-Patients to the Hospital,
and Pathologist, London County Asylums.

LADIES AND GENTLEMEN,—I have brought up to-day for inspection some rather unusual cases of tabes. The usual symptoms of tabes are of course the characteristic ataxic gait, the absence of knee-jerks, the presence of lightning pains, and Romberg's symptom, namely, inability to stand with the eyes shut or in the dark. The cases I shall show you to-day either present symptoms which are not usually met with in tabes, or else there is an absence in them of symptoms which are usually present in the disease. I think it may be considered as proved that tabes, in the majority of instances, is due to the effect of syphilis. I do not say that we should consider tabes to be a syphilitic disease, nor a disease which pathologically is very closely allied to tabes, namely, general paralysis of the insane; but I regard them both as being the result of the action of the syphilitic poison many years before. Of course it might be argued that cases do occur in which there is no history of syphilis at all, though that does not in the least stand against what I have said, because, as I have already mentioned, I do not regard either tabes or general paralysis as syphilitic diseases, but they are the effects of the poison producing primary and progressive decay of systems of neurons. Tabes, speaking generally, is a disease of that portion of the nervous system which is concerned in conducting afferent impressions, because, as we know, in tabes you have no loss of muscular power, but you have muscular inco-ordination, and the disease is for the most part in the posterior roots, the posterior columns

of the cord, and the peripheral sensory nerves. General paralysis is a disease of the nervous structures of the brain, especially of the neurons of association, but *often in general paralysis you have tabetic symptoms*, in fact the tabetic form of general paralysis is not uncommon, and occasionally you find cases of tabes which go on afterwards to general paralysis. Therefore I think it may be considered that these two diseases have a close resemblance ætiologically and pathologically.

Now if tabes is a primary degeneration of the nervous structures which conduct afferent impressions to the central nervous system, we can understand how many varied symptoms you may get in this disease. For example, you may have a case in which an early symptom is blindness. I will show you an instance of that.

CASE 1.—This patient came to my out-patient department from the Westminster Ophthalmic Hospital. He first became blind in one eye, and then in the other. When we examined his discs we found that he had *grey atrophy*, which is a condition characteristic of tabes. That is to say the discs have no longer a rosy tint, but an appearance like mother-of-pearl or a white surface when lightly rubbed over with lead pencil, and the vessels are normal in size. The second eye was affected very soon after the first. A little while after that he was seized with *lightning pains* in the legs. I should mention that he had a *squint*, which is very common as an early symptom of tabes, and in this case it came on before the blindness. The patient had *syphilis ten years ago*. The *lightning pains* and the eye symptoms showed that he was suffering from tabes, but the curious part was that *he had exaggerated knee-jerk on the left side, and diminished knee-jerk on the right side*. Therefore I told the students that next time he came we should probably find the knee-jerks would be absent altogether, because of the lightning pains, evidence doubtless of irritation and subsequent degeneration of nerve-roots. A fortnight later, when he next came, the knee-jerks were both absent, as you see they now are. It is very curious that these people who suffer with grey atrophy of the discs very often remain in the *pre-ataxic stage for a long time*—many years. That is to say, although they have had the lightning pains and the absence of knee-jerks and this disease of the optic discs, yet they are able to

walk about for some time afterwards. Although blind, they yet do not stagger or have difficulty in preserving their equilibrium. I had one patient whom I showed to a previous post-graduate class who *remained in the pre-ataxic stage* twenty years, and then he developed a marked ataxic condition and died two years later. So that although this man is blind, one can give him a little hope by saying that perhaps *he will not get into the miserable paralytic condition so soon as an ordinary case of tabes*. This patient, as you see, can stand well even with his heels together, notwithstanding his blindness. He has lately had symptoms pointing to *laryngeal crises*, for he tells us that he is suddenly seized with spasms and difficulty of breathing; it is rather an unusual symptom in tabes. With regard to the disappearance of the knee-jerks while he has been under observation, I may remark that unless the fourth and fifth lumbar nerve-roots are affected you will not get the absence of knee-jerk, and probably the disease had not attacked these particular roots when he came three weeks ago. The lightning pains which he then had were probably the first indications of the roots being affected. Therefore the *interest about this case is the disappearance of the knee-jerks while he has been under observation, the existence of the grey atrophy*, and the fact that he is able to stand quite well with his heels together notwithstanding his blindness; in fact, you would not tell from his walk that he was tabetic.

CASE 2.—You see a very different picture with this next patient, who has got the typical inco-ordination. He walks with a wide base so as to get his centre of gravity within the base, and he has to use a stick. If I ask him to walk while looking up to the ceiling his inco-ordination becomes very marked indeed, for he depends so much upon vision for guiding sensations of the position of his feet and legs, that if we take away the visual guide he is unable to walk. You will also observe that he cannot stand with his eyes shut. The first patient I showed you was blind, and yet he could stand properly. This patient, who has the ataxy so marked, was at one time a champion hurdle racer, he could do 120 yards with hurdles in most remarkable time. He has also been a mounted policeman. He had to sit for a long time at a stretch on horseback, and the first symptom that he noticed was *shooting*

pains in the left arm. At that time I did not know that he was a *mounted* policeman, and was at a loss to account for the pains in his arm rather than his legs, knowing that pains generally start in the lower limbs. But the man made the very shrewd remark that he was a *mounted* policeman, and held the reins a great deal, and his arms—particularly the left, with which he held the reins—were exposed very much to the weather. I think that is the probable explanation. You will notice that he has *marked inco-ordination in the arms* as well as in the legs; I will show you his writing by asking him to write in this book. You will observe there is *marked inco-ordination*, and he cannot hold the pen properly. Now if I ask him to *write with his eyes shut* he does not even know how to keep his pen on the paper, and has a tendency to go up to the far right-hand corner. So marked is the inco-ordination that he cannot button his clothes now. He has got *Argyll-Robertson pupils*. This phenomenon is present in more than 80 per cent. of the cases of tabes; it seems even a more valuable diagnostic symptom than absent knee-jerks. He has a feeling like wool on the soles of his feet, indicative of *impairment of cutaneous sensibility*. He has had the *girdle sensation*, but that has now disappeared, and he has still shooting pains in his hands and arms. I gave him antipyrin for the relief of the lightning pains, with excellent results. He has not the sense of position of his joints, because he cannot with his eyes shut put one hand into the same position as I put the other. If you were to lay him on a couch and tell him to bend his knee, you would find that he had very fair strength in his muscles, and that they react to galvanic currents. Fifty years ago, when these cases were all called paraplegia, Todd pointed out that it was a disease of the posterior columns of the cord, and that it was muscular inco-ordination which caused loss of the use of the legs, and not loss of muscular power. The patient tells me his knee-joint is swollen. Joint swellings come on in tabes sometimes, and I will show you shortly a very typical case of arthropathy of tabetic origin, what is called Charcot's joint, and very often you get spontaneous dislocation occurring in these cases.

CASE 3 is of interest chiefly because it presents *Charcot's joint*, otherwise there are the characteristic phenomena of tabes—that is to say, he has got the Argyll-Robertson pupils, tabetic gait, light-

ning pains, absence of knee-jerk, inability to stand with his eyes shut, but you also see that *the right ankle is enormously swollen*, but even now it is not so much swollen as it was. The *swelling is quite painless*, and it came on *suddenly*, which is also a characteristic feature of the disease. He was not taken bad until the second week in September, so you see the joint affection has come on very early in the disease. He has not had any attacks of sickness—that is to say, no *gastric crises*. The patient has not got Romberg's sign very marked, but when it is present only in slight degree a severe test is to cause the patient to stand on one foot with his eyes shut.

CASE 4.—The next patient is a clerk aged 44. As there are some points in his case which I would prefer not to allude to before the patient, I will read a few of the notes of his case when he first came to the hospital four months ago. There is a history of syphilis fourteen years ago. He complains of *lightning pains*, particularly in the legs, since he had influenza; he also complained of *difficulty of walking in the dark*. He first became aware of his unsteadiness on washing his face. The pupils are *small*, and present the *Argyll-Robertson phenomenon*. The knee-jerks are exaggerated on both sides, the tongue is protruded in the middle line; there is no tremor, his grasp with the right hand is distinctly less than with the left. He complains of *pains in his arms*, which, together with *rectal paroxysmal pains*, prevent him from sleeping. His memory is also failing. Owing to the severe pain he suffers he often feels as if he would like to commit suicide. He complains of a *pain round the head*, like that produced by a tight hat. There is *nothing characteristic in his speech*, but he is easily upset by trifles. The patient said he felt he could kill a child whom he heard running up and down the corridor in the out-patient room. He says he has rectal pains once or twice a week, and that when they come on they last all night—*rectal crises*. He has slight loss of sense of position, and he fails to touch the tip of his nose accurately with his eyes shut. There is no loss of sensation, but the gait is ataxic. A little later it was noticed that there was slight weakness on protruding the tongue. The rectal pains had left him, and he had not the suicidal tendency. On February 23rd the knee-jerks were still exaggerated, he had the Argyll-Robertson phenomenon, and he could

not stand with his eyes shut. [The patient was now brought in for examination.] He tells me that he has had double vision, due, no doubt, to a *slight squint*, but it has now passed off. One of the commonest things which brings an early case of tabes to the hospital is either *double vision* or a *drooping of the eyelid*, which is very often transitory. On inquiry I find the bruises on his face were caused by a fall. He says that the lightning pains seized him in the legs so suddenly as to cause him to fall down in the road. There are facts which I have narrated in this man's case which suggest that this may have been a *slight seizure* of general paralysis. His ataxic gait is more noticeable than it was formerly, and he walks with a wider base. You will notice that when I ask him to walk while his eyes are directed to the ceiling he falls against the table. He cannot stand if he puts his heels together and closes his eyes. You will notice that his knee-jerks are very much exaggerated, showing that he has some sclerosis in his lateral columns as well as in the posterior columns. The ataxy and the Argyll-Robertson pupils have come on within the last six months. Earlier he had a difficulty in using his hands, and you will notice he goes very wide of the mark in purposive movements, such as touching the tip of his nose with the eyes shut. The man noticed his double vision in a peculiar way, namely, by noticing that the Venetian blinds were not horizontal. He shows symptoms of mental disease, for he tells us that a man has been walking by his side wherever he went trying to persuade him that rooks were owls, and that he told this to his doctor, who managed to persuade him that it was all wrong, and that it was only an hallucination, but he "thinks there is something in it." Here is an instance, therefore, of a man feeling that it is unreal, and bringing all his reasoning powers to bear to try and persuade himself that it is so, and yet he cannot altogether shake off the impression. Still there is no dementia, and that is almost a fundamental condition before you can diagnose general paralysis. The hallucinations this patient has, may however, cause him to become dangerous at any time, and he should be carefully watched. He is under the care of a medical man, and his friends have been warned of the danger.

CASE 5.—This man was in the army for some time, and has had syphilis. He has got absence of

knee-jerks, pains in the legs, girdle sensation, unsteadiness in a very marked degree. If I tell him to raise himself on his toes and then go back again he cannot do it; instead of going back he has a tendency to fall forward. I meant to have pointed out in one of these patients a *loss of tone in the muscles*. I believe it plays an important part in tabes. The muscles are not equally braced up to their proper tonic contraction, owing to the diminution or absence of peripheral stimuli which come from the soles of the feet, and from the tendons, joints, and muscles. This can be shown by laying the patient flat on his back, and while one leg is resting on the bed with the knee extended, the other (with the knee also extended) is raised up as far as possible. In a normal person, owing to the tonus in the hamstring muscles, you cannot raise it much more than to an angle of 120° with the body before it becomes painful; but in a tabetic patient one can often get it up to a right angle owing to loss of tonus of the hamstring muscles which are put on the stretch in this experiment. There is no doubt, I think, that unequal reflex spinal tonus in the muscles explains, in a measure, the inco-ordination of movement in tabes. The case of the patient before you is, however, interesting, because one of the earliest symptoms was *attacks of gastric pain—gastric crises*. He is sick even without food in his stomach. Twelve years ago he had syphilis, and you will notice that the tabetic symptoms usually date from about ten to fifteen years after an attack of syphilis. The first symptom this patient noticed was bladder trouble, so that he has also had vesical crises. When the patient had the pains in his limbs he thought it was rheumatism, and that is usually the case, patients believe they have either rheumatism or sciatica.

People who are not adequately treated are more likely to be affected by diseases like general paralysis and tabes, than those who are thoroughly treated; and a fact which has been pointed out by many neurologists is that people who have very mild attacks of syphilis (perhaps they had a sore which is considered to be a soft sore, and treated perhaps inadequately) are more liable to develop tabes than other cases. I have seen, however, bad cases which were treated thoroughly over a long period, and yet they had tabes.

Women are certainly less subject to tabes than

men; one reason for that, I should think, is that they are less liable to contract syphilis. Still I do not think that accounts for the degree of preponderance. I believe that for every case you see in a woman you will see twenty in men; the disproportion in cases of syphilis is not so great as that. I believe syphilis is on the increase in women. As to the connection between syphilis and tabes, this disease is almost unknown where syphilis is practically unknown. Some people think that tabes is more likely to develop in people who use their legs a great deal, such, for instance, as postmen, policemen, officers; that is, they use the structures most which are most depressed in vitality by the poison, whatever it may be. Exposure to cold and damp would be an additional predisposing cause, so also is injury, for there are cases recorded where injury has been followed by tabes. All four limbs may be affected in the disease; usually in the legs it is the ordinary lumbosacral tabes; or you may have dorsal tabes, or cervical tabes, or cervical bulbar tabes—that is to say, having bulbar symptoms right from the beginning. These latter cases are much rarer than the others. You may have cases in which dementia is an early symptom, because the degeneration has attacked the neurons of the brain as well as the posterior spinal ganglion. The modern view of tabes is that it is a degeneration of the neurons of the posterior spinal ganglia, the processes of which extend by the posterior roots into the posterior columns of the cord. These five cases show that different portions of the afferent projection system of neurons may be affected. In one it is the sense of vision, in another the afferent system of the lower limbs, in another of the upper limbs, while in another it is visceral. The symptoms which arise depend entirely upon the system of neurons which are the seat of the morbid process. The sclerosis of the posterior columns of the cord is not primary, but secondary to the degeneration and atrophy of the fibres caused by trophic changes in the neurons of the posterior spinal ganglia.

A SEVEN-MONTHS-OLD infant, apparently dead from suffocation in croup, was revived by intubation, requiring nine insertions of the tube in twenty-two days, the tube being in the throat a total of 390 hours.

Journ. Amer. Med. Assoc., June 11th, 1898.

A CLINICAL LECTURE DELIVERED AT GUY'S HOSPITAL,

May 10th, 1898,

By DR. J. W. WASHBOURN.

GENTLEMEN,—Of the cases I am bringing before you to-day, one is an undoubted and the other a doubtful case of infective endocarditis. I will begin by reading you the notes of the undoubted case.

The patient is a girl *æt.* 14, who was admitted into the Clinical Ward on April 2nd of this year.

It appears that her father had been a patient in the hospital with rheumatism, but with this exception there is nothing of importance in the family history.

She herself was in the hospital on two occasions—in 1893 and again last year, both times suffering from rheumatism.

The present illness began fourteen days ago, when she complained of shortness of breath and of pain in the left side of the chest. She attended the hospital and was relieved by treatment. Four days ago she was attacked by pain in the back and in the left hip, and two days later spots came out on the legs.

On admission the temperature was 101.2° , the pulse 100, and the respirations 24. She complained of pain in the left side of the chest and back, but there was no pain localised in any of the joints. There were numerous purpuric patches on the legs and ankles. The cardiac dulness was a little increased to the left, but the apex-beat was in the normal position. The impulse was forcible and heaving in character, and there was a loud systolic bruit at the apex, traceable into the axilla and audible at the angle of the scapula. The second sound in the pulmonary area was accentuated. The respiratory system was normal. The urine was normal: it was of the usual specific gravity, and contained no albumin.

Under treatment with salicylate of soda the temperature came down and she got better, but she still complained of pain in the back. On April 6th (four days after admission) there was a good deal of pain in the back and across the loins. The temperature was 100° during the night. Urine normal. On April 13th (eleven days after admis-

sion) the note says that the pain in the lumbar region had continued. There was not any pain on pressure or on movement, but the third and fourth lumbar spines appeared prominent, and there was some rigidity in that part of the back. On the 17th she had great difficulty in retaining her motions, which she passed under her several times. There was no difficulty with micturition. She vomited several times. On the 19th the note states that she still passed her motions under her, but that she was quite conscious, and there was no question of the incontinence of *fæces* being due to her general condition. On the 23rd she got better of the pain in the back and could sit up, and by that time the trouble with her motions had disappeared. On April 25th there is a note to the effect that she was becoming much more anæmic. On May 1st (about a month after admission) blood appeared in the urine. The pains in the back had disappeared, and there was only slight uneasiness in the cardiac area. The spleen was slightly enlarged. On May 4th she had a rigor, and on the same day she vomited, the vomiting being probably caused by methylene blue, which she was taking. On May 7th a bacteriological examination of the blood was made, but no bacteria were discovered. Only a small quantity of blood was examined.

The chart shows that ever since admission she has had some pyrexia, the temperature varying from 99° to 103° .

These are the notes of the case. Now let us consider any points of interest which may occur to us.

With regard to the family history, her father was in this hospital with rheumatism. Patients who suffer from rheumatism generally have a history of rheumatism in the family. The disease is undoubtedly hereditary, and it is interesting to consider what we mean by heredity in disease. There are two kinds of heredity in disease—either the disease itself, or the predisposition to the disease may be inherited. For instance, in syphilis the actual germs of the disease are transferred from the parent to the child. In tubercle the predisposition to the disease and not the actual bacilli is transmitted. People with a predisposition to tubercle do not get tuberculosis unless they are exposed to contagion, but if they are exposed they are more liable to be affected than others.

Rheumatism is no doubt a disease due to a micro-organism, although the particular micro-organism has not yet been discovered; and it would seem that it is a predisposition to rheumatism, and not the disease itself, which is inherited.

Our patient had previously suffered from two attacks of rheumatism. Now, most infective diseases are not liable to recurrence: a patient who has had one attack of scarlet fever is less liable to be attacked again; a patient who has once had smallpox hardly ever has a recurrence. One attack confers immunity, consequently second attacks are rare. Now in rheumatism the question arises whether second attacks are due to second infections or are simply recrudescences. It is possible that the rheumatic virus may remain in the body after an acute attack subsides; it may be locked up in some of the cells of the body, and when the resistance of the individual is diminished by some cause or other, the virus is set free, and again starting into activity, produces another attack of the disease. Such an event occurs in malaria, which is a disease due to a definite micro-organism of an amoeboid form. During the intervals between the attacks these micro-organisms are locked up in various tissues, such as the spleen and liver, and when the patient gets run down in health they escape into the blood, and so produce a recrudescence of the fever. The proof of this is that patients who have had malaria may again be attacked after removal to districts where there is no possibility of reinfection from without. But second attacks of rheumatism may be due to fresh infection. We know that persons who have suffered from influenza are quite as liable, if not more liable, than other patients to contract the disease again. Many people suffer from influenza in every epidemic of the disease which prevails. Some infective diseases confer a very transient immunity, so that after a brief interval the patient is as liable as any one else to another attack.

Now with regard to the condition of our patient when first seen, the principal points are these:—just before admission she had pain in the back and hip, probably of a rheumatic nature taking into consideration the previous history of the case. On admission she undoubtedly had mitral regurgitation. There was a loud systolic bruit at the apex traceable into the axilla, and an accentuated second sound in the pulmonary region.

When these signs are present you may be satisfied that there is mitral regurgitation. An apical systolic bruit may be functional or hæmic; but when it is traceable into the axilla, and the pulmonary second sound is accentuated, you may be sure there is mitral regurgitation. An accentuated second sound in the pulmonary region is of great importance; it means that there is high tension in the pulmonary artery, which would be the natural result of mitral regurgitation. During the systole of the heart the blood is forced from the left ventricle through the mitral valve into the left auricle; during the diastole there would be an increased tension on the pulmonary side of the circulation, and the pulmonary valve would be shut with a great deal of force, and so produce a loud sound. This is an important symptom of mitral regurgitation, and often is of great assistance to you in determining whether an apical bruit is due to mitral regurgitation.

In addition to mitral regurgitation, our patient suffered from pyrexia, from pains in the back and hips, and from petechiæ on the legs. The pyrexia and pain might have been due to rheumatism, and people with mitral regurgitation, if they walk about, are liable to get petechial extravasation on the legs. There was, however, a suspicion that the petechiæ might be due to some septic trouble, such as is found in infective endocarditis; but taking everything into consideration,—the previous attacks of rheumatism, the pains in the back and hip, and the pyrexia,—the conclusion was that she was simply suffering from another attack of rheumatism, and that the cardiac trouble was due to the previous attacks.

But later on we find that, although the pain in the hip disappeared, the pain in the back remained; in fact it became worse, and that in spite of the administration of salicylate of soda. Then she began to suffer from incontinence of fæces. Now, in an attack of rheumatism, incontinence of fæces is exceedingly rare, and is only met with when the patient is delirious or comatose. Our patient was not in that condition, her mental condition was good, and she did not appear to be very seriously ill. The pain in the back and the incontinence of fæces suggested caries of the spine; but there was neither paralysis of the limbs, nor loss of sensation, nor incontinence of urine. A diffuse pressure caused by caries would hardly account for a

paralysis localised to the centre for defæcation in the lumbar region of the cord. A more probable explanation, which was subsequently adopted, was that of embolism of a branch of the spinal artery supplying the part of the cord in which the centre for defæcation is situated.

Later on other symptoms developed; blood appeared in the urine, and the spleen became enlarged. Now these are very important symptoms when they arise in the course of heart disease. Blood in the urine may be produced through a simple non-infective embolism, and so may an enlargement of the spleen. Where there is mitral regurgitation a little fibrin may be detached from the valve, or a clot in the auricle may be drawn into the circulation and thus produce an embolism. If such an embolus were to get into the kidney it would very likely give rise to the passage of blood in the urine.

A little later it was found that the pyrexia continued, and that there were rigors in addition. When you take all these symptoms together there can be no doubt that we were dealing with a case of infective endocarditis. That there was an affection of the valves was shown by the mitral regurgitation and by the occurrence of embolism. The continuance of pyrexia in spite of treatment proved that the pyrexia could not be due to rheumatism; we were therefore forced to look upon it as of septic origin, and no doubt the acute nephritis and the petechiæ on the legs were of a similar nature. Probably the embolism in the spinal cord was non-infective, for the spinal trouble quickly subsided, and did not pass on to an acute inflammation.

You will remember I told you a bacteriological examination of the blood was made, but that micro-organisms were not found. I shall discuss this question later; but it will be convenient first to read you brief notes of the second case, in which there is a suspicion only of infective endocarditis.

This was also the case of a girl of fourteen years of age, who was admitted into the hospital for dyspnoea and pains over the heart and liver. Her mother suffers from rheumatism, and she has eight brothers and sisters, most of whom have had rheumatism. She herself had rheumatism in 1896, and was a patient in this hospital in March of this year with mitral and tricuspid regurgitation. She left the hospital on March 30th, and since that

time she has again suffered from cardiac distress, and from pain in the region of the liver, and cough.

On admission she was very dyspnoic, the lips were cyanosed, there was clubbing of fingers, and slight œdema of the feet. In the notes there is a detailed account of the condition of the heart, which practically comes to this, that the heart was enlarged, that there was a loud systolic bruit at the apex, that the second sound at the apex was followed by a feeble diastolic murmur, and that there was a good deal of pulsation in the third intercostal space on the left side. The liver was considerably enlarged, and the splenic dulness was increased, although the spleen could not be felt. There were râles and rhonchi in the chest.

She has continued since admission much in the same condition, but has become more anæmic, and a very loud systolic bruit accompanied by a distinct thrill has appeared in the pulmonary area. The temperature has remained a little above the normal except for the first two or three days after admission.

These are the main points in connection with the case:—there was a history of rheumatism in the family, and there were previous attacks, in the second of which the heart was distinctly affected, possibly before, but we have no notes of the first attack. On admission the cardiac symptoms were pronounced, and there was mitral regurgitation and stenosis, the evidence of stenosis being the diastolic bruit following the second sound. The pyrexia has continued, and there has been a change in the character of the cardiac bruits. For these reasons there is a suspicion of infective endocarditis. Her anæmic condition can, however, quite well account for the bruit to be heard in the pulmonary area, and the pyrexia may be due to the rheumatic condition.

Now let us consider the chief points in connection with endocarditis. Endocarditis may be of two kinds, acute or chronic. Chronic endocarditis may be a primary condition due to such diseases as gout or syphilis, but it is more frequently consequential to an acute endocarditis. Although endocarditis may attack any part of the endocardium, it affects the valves more than any other part. In chronic endocarditis the valves become thickened, causing symptoms of valvular incompetence, a subject we cannot now discuss. Acute endocarditis may be of two kinds, simple or

infective; the real and essential difference between these two conditions is that in the simple form no micro-organisms are found on the valves, while in infective endocarditis various bacteria are present. Simple endocarditis usually affects the left side of the heart, and is almost always due to acute rheumatism or to chorea which are manifestations of the same condition. Small vegetations form on the valves, which microscopically are found to consist of a round-celled infiltration. The tissue on the surface of the valve undergoes coagulative necrosis, and, upon the surface of this necrosed tissue, fibrin is deposited from the blood. It is quite possible that some special micro-organism is present on the affected valve, but nothing can be detected with the methods now available. In consequence of the change in the shape of the valve there may be valvular incompetency, causing symptoms of mitral or aortic obstruction and regurgitation. After a time this acute inflammation may go on to a chronic condition, so that the valves become sclerosed, producing all the symptoms of valvular disease. The vegetations sometimes become atheromatous, and then atheromatous ulcers form upon the valve similar to atheromatous ulcers in any other part of the arterial system, which shows that the term ulcerative endocarditis is not a good one to use in place of infective endocarditis. In simple endocarditis ulceration may occur, but the condition is not that of infective endocarditis.

Part of the valve or a little fibrin may become detached, and, getting into one of the vessels of the brain, may produce paralysis, or getting into the spleen or the kidney may produce an infarct. But this does not give rise to suppuration, because the emboli which are brought into the vessels do not contain organisms which cause this condition. If there are organisms in the simple form of endocarditis, they do not give rise to suppuration, the effect is much the same as if a piece of sterile tissue were injected into the circulation.

In infective endocarditis, however, a different condition arises, for bacteria are present upon the vegetations. Those most frequently found are the streptococcus pyogenes, the staphylococcus pyogenes, and the pneumococcus. Other bacteria, such as the tubercle bacillus, the typhoid bacillus, the anthrax bacillus, and the gonococcus are sometimes found.

How do these micro-organisms enter the body?

It is sometimes an easy matter to trace out the path of infection. In pneumonia the pneumococcus grows in the lungs, from whence it may get into the circulation, and settling upon the valves of the heart, may produce a pneumococcic endocarditis; in puerperal fever the streptococci growing in the peritoneum may get into the circulation and so produce a streptococcic endocarditis; and the same thing may occur in septicæmia and in erysipelas. In gonorrhœa the gonococcus may escape from the urethral membrane into the blood, and so produce a gonococcic endocarditis. We thus find that infective endocarditis is frequently a complication of some bacterial disease, such as pneumonia or septicæmia. But you must remember that there are other cases in which infective endocarditis arises without there being the slightest evidence of the mode of entrance of the virus. For instance, there was a case in Mary Ward last year of a girl who had a very marked attack of infective endocarditis, yet the most careful examination failed to reveal any evidence of a portal by which the infection could have entered. In that case we thought of the possibility of the virus entering through an ulcer in the intestine or through the lungs without giving rise to any inflammation at the point of inoculation. It is possible for a pathogenic bacterium to enter the body without leaving any trace of the path by which it came. In acute osteomyelitis pathogenic cocci are present in the bones, but we do not know how they enter the body. If they enter through the lungs they leave behind no evidence of their track.

Why do the bacteria settle upon the valves? In some cases the reason is that the valves have been previously injured, and when a valve is diseased there is a local predisposition of that part to infection. On the other hand, valves previously quite healthy may be the seat of infective endocarditis. In such cases we can only suppose that there is a general predisposition of the patient to infection.

As far as experimental work is concerned, if certain pathogenic micro-organisms are injected into the blood after mechanical injury to the valves, infective endocarditis is produced.

The morbid anatomy of infective endocarditis is somewhat different to that of simple endocarditis. In the simple form, the vegetations ultimately become sclerosed and converted into fibrous tissue, or they become atheromatous. In infective endo-

carditis the vegetations show no tendency to be converted into fibrous tissue, but they readily necrose and ulcerate. It is this ulceration which has given to the infective form the name of ulcerative endocarditis, but it is an inconvenient name, because, as I have shown you, ulceration may occur in the simple form of the disease.

The emboli which occur in the infective form are different to those which occur in the simple form. The majority of the emboli in the infective form contain micro-organisms, and wherever these emboli lodge they produce inflammation. If an embolus passes into the lungs it produces suppuration, so that the lungs may become riddled with abscesses; and a similar state of things may obtain in the other tissues. In the kidneys minute septic emboli may cause an acute hæmorrhagic nephritis. If an embolus gets into one of the systemic blood-vessels, the vessel is at first blocked, and then its wall becomes inflamed and softened, and an acute aneurysm results.

It is important in these cases to make a bacteriological examination of the blood, because by that means you may be able to detect what particular organism is producing the disease. It is not of the least good examining a single drop of blood; there are never sufficient bacteria in the blood to render the examination of such a small quantity of value. You must either examine a large quantity of blood taken on one occasion, or you must examine a smaller quantity taken on several occasions. A large quantity can be obtained from the arm with an ordinary syringe, such as that used for injecting antitoxin. The skin is sterilised, and the needle of the syringe inserted into one of the veins at the bend of the elbow, having first tied a bandage above the vein so as to make it prominent. By this means you can draw off a large syringe-full of blood, ten or fifteen cubic centimetres, with the greatest ease. To obtain smaller quantities of blood it is well to employ a sterile pipette such as that used for the serum test for typhoid fever. The lobule of the ear is carefully sterilised with lysol, and afterwards with alcohol, the skin is pricked with a sterilised needle, and the blood is drawn up into the pipette. The ends of the pipette are sealed in the flame, and the blood conveyed to the laboratory for examination. By this method a cubic centimetre of blood can be obtained, and it will usually be

necessary to make a number of examinations before obtaining a positive result. In fact, in many cases, even when large quantities of blood are examined, no bacteria can be detected.

With regard to the symptoms of infective endocarditis, two distinct conditions must be remembered: first, valvular incompetency, caused by the mechanical condition of the valves; secondly, a septicæmia produced by the bacteria; sometimes the mechanical effects and sometimes the septic effects are most marked. Sometimes the symptoms are entirely those of acute septicæmia without any evidence of cardiac affection, and yet after death vegetations containing bacteria are found on the valves. In other cases, like the second one I have read you, the principal symptoms are cardiac—enlargement of the heart, œdema, dyspnoea, &c.—and it is doubtful whether there be some infective trouble in addition. Often the only reason for suspecting infective trouble is continued pyrexia or a change in the character of the cardiac bruits.

With regard to the septic symptoms, various types are observed. There may be a typhoid condition, with coma, collapse, high temperature, and profuse sweating; or there may be acute septicæmic symptoms, with frequent rigors, irregular temperature, sweating, and wasting; or the embolic processes may be most marked with symptoms similar to those of pyæmia and with acute aneurysms in various parts of the body.

With regard to treatment, the patient must be placed in the very best condition for recovery; he must be well fed and well nursed, and must be treated either as a cardiac case or as a case of septicæmia, according to the symptoms. The only specific treatment is that by serum. It is with a view to treatment of this kind that a bacteriological examination of the blood is made. If streptococci are found you may be assured that they are the cause of the septicæmia, and in such cases treatment with antistreptococcic serum is often of the very greatest value. In some cases it does not seem to do much good, but in other cases I am sure it has saved the patient's life. But the serum treatment cannot be adopted in all cases—for instance, in cases of gonococcic endocarditis—because there is no serum at present available for that purpose. And it is not of much use giving the serum in those cases where the cardiac sym-

ptoms are very prominent. In one case of this nature I believe the serum did remove the infective process, but the patient shortly died from the cardiac affection. In such cases the treatment must be similar to that of ordinary valvular incompetency.

The prognosis of infective endocarditis is always serious. When the infective process is implanted upon a heart already the seat of severe valvular disease the prospect of recovery is very slight, but when the valvular trouble is not very marked the prospect is better, especially if serum treatment can be adopted.

Mechanical Treatment of Obesity.—Muscular activity, that has been so widely advised, is not always good, on account of the hyperthermia that this activity produces. In noticing the change in temperature in cases in which obese patients have undergone violent exercise, we find that it is sometimes of a febrile character, and that the organic albumen loses its vitality. This process consequently causes nervous debility, anæmia, and albuminuria. It is necessary to invent a means of increasing oxidation, and at the same time avoid hyperthermia in such a way as to destroy the fat without affecting the albumen. To this end Winternitz advises lowering the temperature of the body before exercise. It is easy to do this by means of a bath of 12° or 14° C., lasting fifteen or twenty minutes. By this means one can take away five hundred calories from the organism. To recuperate these fifty grams of fat must be consumed. In order that lowering of the temperature may not cause contraction of the cutaneous vessels, he proceeds as follows:—In the morning submit the patient to an energetic rubbing with a towel wet in cold water; then place him for five or ten minutes in a steam room; on leaving this room he takes a bath at a low temperature, lasting from three to six minutes. Severe muscular exercise terminates the treatment. This may be repeated two or three times in twenty-four hours. It is useless with this method to prescribe a rigid diet; the results are rapid, and are never accompanied by troubles similar to those observed with the other methods.

Medical Record, June 11th, 1898.

CHAPTERS FROM THE TEACHING OF DR. G. V. POORE.

No. II.

GENTLEMEN,—The reason why hearsay is not evidence, and why dying declarations, which are forms of hearsay, are received with great unwillingness, is this: They cannot be subjected to cross-examination, and cross-examination is the very essence of our judicial system. Evidence is almost worthless unless it be subjected to cross-examination—

"A lie which is half a truth is ever the blackest of lies,"

and that is why cross-examination is so necessary. A man may state facts quite truthfully, but the way in which he puts them may give a totally false impression. I once heard it stated of a man that "he lived down a blind alley out of Oxford Street." It was quite true, but on cross-examination it appeared that he lived in a palatial residence in Stratford Place, which happens to be a *cul-de-sac*. Here the use of cross-examination comes in. Now the cross-examination is made by a hostile person, and it is the business of the cross-examiner to shake your evidence and to turn it, if he can, in favour of his own side. A cross-examiner, as I have said, is allowed to put leading questions, and he has been known to bully. You must not mind that, and it is a very great mistake for a witness, especially a scientific witness, to lose his temper or to bandy words with the counsel. If you speak the absolute truth you need fear neither examination nor cross-examination. It is when a man tries to give a biassed view, when he tries to appear learned about a thing that he really knows little or nothing about, that the cross-examination comes to him as a trial. If you are perfectly truthful and simple, and, let me say, think of your own reputation, not only for knowledge, but for honesty and truthfulness, you need not fear any cross-examination.

"This above all, to thine own self be true,
And it must follow as the night the day,
Thou canst not then be false to any man."

And that is the mental condition a man should be in when he gets into the witness-box.

Now cross-examinations are made by persons of

great ability, and you must remember that the eminent counsel is a man of peculiar mental organisation, who acquires knowledge without the slightest difficulty. He can acquire knowledge of every kind; be perfectly clear-headed and be able to cross-examine on it. If you doubt this, read the trial of Madeline Smith or William Palmer for murder. There you will see what Inglis in Edinburgh, or Cockburn in England, could acquire in the way of a knowledge of chemistry and the detection of poisons. Remember you have to face a man not only of great ability, but a man who has got books and experts behind him, telling him what questions should be asked, and why.

May one quote authorities? Everything that is in print acquires an importance which very often it ought not to have, and there is no doubt whatever that authorities are calculated to influence the jury. But my advice to you is never to quote them. And you must remember that authorities come in the same category as hearsay; the author cannot be cross examined. You may quote from Taylor or Christison, but you cannot call them from the vasty deep and cross-examine them. And you must remember that a clever advocate has possibly a more accurate knowledge of authorities than you have yourself, because if he is going to cross-examine you upon a technical point he has probably got all the authorities at his fingers' ends. That is the quality which brings a man to the front at the Bar. To give you an instance, at the trial of Madeline Smith in Edinburgh the question arose whether white arsenic had any taste, that is to say, whether a person might take white arsenic unawares. One of the medical witnesses said that arsenic had no taste, that it was merely rough on the tongue, and he quoted a passage from Orfila's book to the effect that white arsenic was "*apre*" (rough). The opposing counsel was down upon him instantly. "Yes, quite right; page so-and-so, volume so-and-so. But on page so-and-so you will find he says it is '*acre et corrosive*.'"

Again, you must remember that you may have an authority quoted at you. Counsel may ask you, "Do not you know that So-and-so, the great authority, gives this opinion?" When an authority is quoted at you, if you do not accurately remember the passage, *always ask to see the passage referred to*. Remember that a passage in a book can only be fairly interpreted by the context. Do not have

an odd paragraph quoted at you without being sure as to what is being quoted. It is a very important thing to keep your head in the witness-box.

In giving evidence, and when under cross-examination, always be respectful to colleagues. It is a very necessary thing. You may give an opinion, and are told that another medical witness has given a contrary opinion. The medical witness may be of no account whatever. You may have the very worst opinion of him, and such an opinion may be justifiable; but do not use any expression which savours of contempt. Do not say it is "rubbish" or "nonsense;" simply say you do not agree with him. I think that is very important.

Do not appear unwilling to communicate facts. If you know anything, say so. Another important point is to keep matters of fact and matters of opinion perfectly distinct. That is not always done.

Now after your cross-examination, if the cross-examining counsel has given a different aspect to anything you may have said than that which you gave it in your examination-in-chief, there is a re-examination by the friendly counsel. He will re-examine you, so as to correct any false impressions which may have been left on the jury. So much about the ways of giving evidence.

You may be called, as I have said, as a common witness to testify to facts; or you may be called as a skilled witness to give your opinion upon facts. The advice on this head which I have to give you will almost appear unnecessary. Do not go as a *skilled witness* unless you are *really* and *practically* conversant with the facts concerning which you go to testify. As a professor of medical jurisprudence, I am very often appealed to by lawyers to give evidence, and I will give you an instance of when I refused to do so.

A year or two ago a lawyer came to me to know if I would give evidence upon a case in which the legitimacy of a child was disputed. It seems that a lady had run away from her husband, and nine months after the day she ran away from her husband a child was born. The question arose as to whether that was the son of the gentleman she left at Basingstoke, or the son of the gentleman she met at Waterloo. And they came to me to give evidence. The point at issue was the relationship of pregnancy to menstruation. Well, I may say this; I have on

my bookshelves dozens of books that deal with the subject, and when I come to deal with it in these lectures I shall give you information which I hope will be useful to you. I know what the *authorities* say upon the matter, but I would not go. Why? Because I felt I could not stand being cross-examined. Supposing the adverse counsel had said to me, You are practising as a physician, I believe? Yes. You are not an obstetric physician? No. Are you in the habit of seeing a large number of obstetric cases? No. When did you last attend a midwifery case? Thirty-two years ago. That would have been my truthful answer. Now, although I could give the lawyer good advice, and did give him what proved to be good advice, it would have been very silly for me to go as a witness. It does not follow that because you are a doctor you know every branch of doctoring, and it does not follow that because I am a professor of medical jurisprudence I have a practical and extraordinary knowledge (which a skilled witness should have) of obstetrics. So I did not go, and that will show you what I think your attitude should be in these matters. If you go to speak about a thing which you really are particularly conversant with, then you will be useful, and you will do credit to yourself.

Now it is the custom in this country to call skilled witnesses on either side; and expert evidence, as a consequence, has an evil reputation. But it answers tolerably well, because there is such a thing as cross-examination, and if the expert has an extravagant opinion, or an opinion based upon ignorance instead of knowledge, a clever counsel and those who are behind him may be relied upon to prick the wind-bag, and show how hollow it is. If you are subpoenaed merely to give opinions as an expert are you bound to go? The answer is doubtful. I will only say you had better go. Some years ago there was an artistic quarrel in the Law Courts, and all the Royal Academicians were subpoenaed *en bloc* on one side. These gentlemen all came to obey the subpoena to give evidence, because they felt it would not be safe to disobey it. But when the lawyer comes to you for your opinion as an expert, he first of all finds out what your opinion is before he calls you; and unless your opinion is useful to him he will not call you.

The next point is this. You are sometimes asked to give evidence as almost a matter of charity. There is no rule without an exception. But I would say Don't. The medical profession does a great deal for charity, I hope it will always continue to do so, but be very careful how you take up litigation as an act of charity. And I would say this, Beware of solicitors' actions. There are black sheep in every profession; there are medical black sheep, and there are legal black sheep, and the legal black sheep are sometimes very black ones. And you must be exceedingly careful not to get linked with a firm of dishonest solicitors who are taking up an action simply for what they can get out of it. The taking up of an action for what they can get out of it, is, I believe, illegal; it constitutes the misdemeanour of "Champerty and maintenance," concerning which you will find a great deal in Warren's novel of 'Ten Thousand a Year.' You are not allowed to support a man in bringing an action if you are to benefit by the result yourself. Some years ago I had a letter from a firm of lawyers of whom I knew nothing, and it was couched in somewhat these terms:—"Dear Sir, '*Smith v. the London Omnibus Company.*' Our client, Mrs. Smith, is bringing an action against the London Omnibus Company for damages sustained in one of their vehicles. As I understand Mrs. Smith is a patient of yours, we should be very much obliged if you would let us have your opinion of the case." Do not ever, under such circumstances, give a written opinion to strangers. What did I do? I answered perfectly truthfully as follows:—"Gentlemen, '*Smith v. the London Omnibus Company.*' I am sorry I have no recollection of your client, Mrs. Smith." A few weeks went by, and I got another letter: "Sir, '*Smith v. the London Omnibus Company.*' As we understand that our client, Mrs. Smith, attended at your out-patient department at University College Hospital yesterday, and as we are further given to understand that you made rather lengthy remarks about her condition to a circle of students, and that you finally ordered her a plaster to go upon the back of her neck, we think that these facts may probably have brought her to your recollection. If such be the case, will you kindly furnish us with your opinion?" To that I replied as follows:—"Gentlemen, '*Smith v. the London*

Omnibus Company.' If you will send a member of your firm, or a responsible clerk, to me during my professional hours, I shall be very happy to discuss this question with him, on the distinct understanding that every interview and every letter is treated as a matter of professional business." That was the last I heard of *Smith v. the London Omnibus Company.* Now I advise you to always take a similar line; never allow yourself to be entrapped into what is known as a solicitor's action. I did not know anything about that action; but *my* course of action I feel sure was right. If you give a written opinion you never know where it will end, or where it will take you. Moreover, you have no right to be dragged out of your house and be put to expense without proper professional remuneration. There have been letters in the papers lately stating that a fee of two guineas asked by a doctor somewhere for giving certificates *apropos* of litigation was an extortion. It was nothing of the kind; it was very proper. It was a moderate and proper fee. There is no rule without an exception; but if a person is able to employ solicitors he is able to pay professional fees, and you must be well satisfied that you are going to do a real act of charity before you link yourself with a case without being properly paid for it.

My experience of solicitors' actions came to me very early indeed. I will tell you about it, because I think it should serve as a warning, and you will see why I lay down this rule. When I was house surgeon in 1866 or 1867 there was amongst the students a man of not very good reputation. One does now and then come across such a man. One day he came into my room with a cut across the back of his hand, a clean cut done with a pane of glass, which laid bare the extensor tendons of the little finger and ring finger. I brought the wound together according to the methods in vogue at that time, put on a splint, and sent him away. The account he gave was that, coming downstairs at a railway station, he slipped on the brass edging of the stairs and put his hand through a window, and so cut it. I heard nothing of him for several months, when he appeared in my room with a subpoena ordering me to attend next day at the assizes at Croydon, where his action against the railway company was to be tried. With the subpoena he gave me two shillings, which was enough

to pay my second-class railway fare to Croydon. That was all he was really bound to give me with the subpoena. I had not seen this man for six months, but he still had his hand in a splint. I was rather annoyed at the circumstance. He said to me, "If it will save you any trouble I will call for you to-morrow morning;" and accordingly at 7.30 a.m. on the morrow I found myself in a four-wheeled cab with the plaintiff. We stopped first of all in the New Road, opposite a big door-plate, and were joined by a sporting gentleman with tight trousers and a bit of straw in his mouth. He was a solicitor licensed to administer oaths. Then we went a little further and picked up a medical witness, old and wicked, who had been on the treadmill for perjury, as I afterwards found out. Next we stopped at Serjeant's Inn and picked up the barrister, a gentleman of stalwart dimensions, with a black eye, burst boots, and rotten clothing. And so to London Bridge Station. There we were met by the second medical witness, who was proprietor of a homœopathic dispensary, and employed sandwich-men to invite clients. I found myself, therefore, in the same boat with a very pretty crew. You will please note how I got there. I got there from no fault of my own; I was bound to be there because I was subpoenaed, and I could not refuse to go. Moreover, they could not help calling me because I knew more than anybody else did about the matter. You must remember also I was little more than a boy. The old medical witness took me by the arm in a fatherly, gentle kind of way, and like Hamlet and the ghost we glided to "a more remote part of the platform;" then taking his left forefinger he drew it completely across the back of his right hand, and giving me a look said, "All the tendons on the back of the hand completely divided." I said, "I beg your pardon, not one of them." The man was a rogue; there was no question about it. Then he began to try to bully me. He said, "How do you account for the stiffness?" I said, "If you keep a man's hand in splints for six months you could not account for anything else." He said, "The tendons must have been divided. I was surgical prizeman at —'s Hospital forty years ago, and I know something about surgical pathology. They must have been divided." I said, "I have taken two surgical prizes at University College this year, and surgical pathology has made some advances since your

time. I am absolutely certain none of the tendons were divided, and that will be my evidence." We went down to Croydon; they were at one side of the carriage and I was at the other, and I was thinking over what line to take. It was a very disagreeable position to be in. I was in the company of—I do not hesitate to say it—four criminals, who were engaged in a conspiracy. The fee to which I was legally entitled (£1 1s.) had not been paid, I felt it probable that they had not got a guinea between them, but I said to the lawyer, "My fee has not been paid, and if I am put into the witness box I shall inform the judge." If your fees have not been paid you must inform the judge of the fact *before you are sworn*. After you are sworn he will make you give evidence, because you have taken your oath to speak the truth and nothing but the truth. In a large number of these solicitors' actions the fees are not paid, and the judge, if informed, will order the fees to be paid. I took this line on the occasion I am relating, not so much for the sake of the guinea, but to make it clear that I was not dipping in the same dish with these rascals. I advise you to take the same course always. The judge luckily was a strong one—the late Lord Bramwell,—and the black-eyed barrister had hardly opened the case when he shut it up, saying there was no cause of action. I tell you the tale because it shows you what a solicitor's action is, and how you may be involved in it, and your reputation be endangered. Be cautious in giving opinions and information to solicitors of whom you know nothing.

The scale of fees for giving medical evidence in the higher courts ranges from one guinea to three guineas. If you have been attending a patient, and you know the facts, you are bound to go for them. But supposing people come to you and ask you to give your opinion upon a case in which you have not been professionally concerned, then you become an expert witness, and the fee is a matter of arrangement with the lawyer. It stands to reason that no man in extensive practice would run the risk of being taken away from that practice and being kept perhaps day after day hanging about a law court for a guinea or three guineas; it would not pay him. Therefore the solicitor has to come to an agreement. Now what should you ask? That will depend very much on the value of your time, and the value of your time depends on the

position in the profession in which you happen to be. The value of the time of the late Sir William Gull, let us say, or of the late Sir Andrew Clark, and the value of the time of an obscure general practitioner are very different. Therefore when you are asked to go as an expert what should you do? My advice is this: You should ask the fee which you would ask of a rich patient whose good will you wished to retain. Now if a man gives a very strong and very one-sided evidence, and in cross-examination it is found that he has had an *exorbitant fee*, then the value of his evidence is diminished immensely by that fact, because it may be made to appear that he is giving his evidence because he has been practically bribed to give it. So that the fee you charge must be one which you can defend conscientiously.

There is another point with regard to evidence which has happened to myself. I had once two subpoenas to attend two different courts, one at Clerkenwell and one at assizes in the country, on the same day. I could not cut myself into two, therefore what was I to do? I think you will be perfectly safe to go to the higher court. In this case the assizes in the country was a higher court than the sessions at Clerkenwell. In these days you would telegraph to the solicitor who had subpoenaed you in the lower court to say that you had been subpoenaed in the higher, and were going. It so happened that I was at Clerkenwell when I got the second subpoena, and I asked the presiding judge. He said, "We will postpone the case in which you are concerned until you have given your evidence at the assizes in the country and have come back again." In such circumstances always appeal to the fountain-head for advice.

Beware of giving evidence as a skilled witness unless you know the *whole* facts. Only a few days ago I was asked to give evidence in a case. The lawyer came to me and put the matter before me in such a way that I was more than half inclined to give evidence. But I adopted the rule which I advise you always to adopt. I said, "Before I give you my answer I should like to read the whole of the depositions as taken before the magistrate." These were left with me; I read them all through, and having read both sides of the question, and not merely listened to the *ex parte* statement of the lawyer engaged on one side, I came to the conclusion that I could not

give the evidence I thought I could, and I accordingly refused. A lawyer will sometimes come to you and put one side, and then in cross-examination something may be said which puts a different complexion on the case altogether.

Now there are one or two things which you may be called upon to do. You may be called upon to take down a "dying declaration." As I said yesterday, a dying declaration is admissible only in criminal cases, and only in cases of homicide where the death of the person making the declaration is the subject of the charge. We will suppose a murdered man wishes to make a dying declaration. In order that this declaration can be admitted, the person must be aware that he is going to die, without hope of recovery. The judges always insist upon that. If you are taking down a dying declaration, take it down in the *ipsissima verba* of the dying person. Do not put your own construction upon it. There is not the slightest reason why you should not ask him what he means by any expression, but you must not, under any circumstances, put a leading question to him, such as "Did you see John Smith?" That would be wrong. Then you must get the dying person to sign it or make his mark on it, and you should attest it yourself with the date, and get a witness, if possible. But if it can be avoided do not take a dying declaration yourself; send for a magistrate. That is much more satisfactory. You may not only have to take a dying declaration, but you may have to make a will. Now here again I would say very strongly: If you are asked to make a will, do not if you can possibly help it. If a person is dying in an out-of-the-way place, and there is nobody else at hand, it is your duty to do it. And if you are called upon to make a will, remember again that you must make no suggestions; you must be sure that the will is a free will, and not made in response to suggestion. I will give you a case in point. A doctor attending an old gentleman made his will for him, or helped him to make it. The doctor suggested that his housekeeper, who had lived with him for many, many years, and had been kind and trustworthy, and so forth, was the proper person to be benefited, as he had no needy relatives of his own. As a result of that advice the old gentleman left the bulk of his property to the housekeeper. No sooner was the old man dead than the doctor married the housekeeper.

The relatives disputed the will, and the court decided that the doctor's advice was a breach of trust, as he was giving advice which was meant to benefit himself. Accordingly the doctor got the housekeeper without the money. In respect of wills, also, though you may make no suggestions, you may ask for an explanation if the meaning of a statement is not clear. Do not attempt to make use of legal phraseology. An important thing, without which no will is valid, is the attestation clause. This clause is to the following intent: "Signed by the testator, A. B., in the presence of us"—there must always be two witnesses to a will—"present at the same time"—the two witnesses must be there together—"who at his request"—the request of the testator—"in his sight and presence, and in the presence of each other, have attested and subscribed the same." When a will is attested there must be three persons present, namely, the testator and two witnesses, and they must be present together. Then all alterations, interlineations, corrections must be initialled by the testator and the witnesses. For instance, to take an extreme case, you cannot leave John a thousand pounds and then scratch *John* out and put *Emily* in unless the alteration is initialled by the testator and the witnesses. Wills should, of course, be made with the most indelible and indestructible materials. Wills are usually written upon parchment; but it is said that a will is valid no matter what it is written on. There is a record of a will written with a bit of chalk on a barn door.

Cancer.—'The Monthly Cyclopædia of Practical Medicine' contains an editorial which reviews the treatment of cancer by interstitial injections of alcohol. It appears from this review that much is to be expected from this method, which has been tried occasionally with success for the last twenty-five years. Over twenty-five years ago Karl Schwalbe, having obtained satisfactory results from the interstitial injection of alcohol in the treatment of benign growths, argued that if alcoholism could give rise to the formation of new connective tissue in the liver, and thereby induce atrophy of the parenchyma, including the vascular supply, malignant tumours should yield to the direct action of alcohol in the same manner. Hasse concluded that injection at the base of a tumour would be sufficient. He claims to have cured fifteen out of eighteen cases of carcinoma.

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HERNIA IN INFANCY.*

BY

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THE forms of hernia met with in children are generally umbilical or inguinal, while femoral hernia is not often seen; indeed Keatinge in his 'Encyclopædia of Diseases of Children' states that femoral hernia is never congenital, so that my remarks will be limited to the consideration of umbilical and inguinal hernia, while the rarer forms such as femoral will be set aside.

Umbilical hernia is of two kinds, either congenital or acquired. There is a third variety of so-called umbilical hernia which is really ventral, inasmuch as protrusion of the intestine takes place not through the umbilicus, but generally at some spot near it, very frequently just above it, very rarely below it. The linea alba is naturally wider above the umbilicus, while below it is partially obliterated by the interlacing fibres of the two recti muscles, and by the pyramidales.

The congenital form of umbilical hernia is always a serious condition, inasmuch as it is something more than a hernia. It is a condition of exomphalos—a portion of the intestine probably not having been at any time in the abdominal cavity.

The acquired form is due to dilatation of the natural umbilical opening.

In either case the condition is readily recognisable, and the symptoms are those of hernia in general. With regard to the treatment of the truly congenital form, early reduction is absolutely essential, for the sloughing and suppurating process by which the umbilical cord separates may extend to the hernial sac, and give rise either to adhesion of the bowel to the sac, or even light up a general peritonitis. If it should be found impossible to reduce a congenital umbilical hernia, an operation should be performed at once to avoid the risks just mentioned, and several cases have

* A paper read before the East Surrey Branch of the British Medical Association.

been operated on with success. The acquired form of umbilical hernia is by no means so serious. It varies in size, is generally sessile, but as the mass grows larger may become somewhat pedunculated, and I have seen them projecting until they are about as large as an orange. The factors at work in the production of an acquired umbilical hernia are much the same as those of other infantile forms of hernia. They are those which increase the pressure in the abdominal cavity, such as cough, phimosis, straining at stool, and many others which will be spoken of more fully presently.

The treatment of an acquired umbilical hernia is very simple, but all forms of apparatus should be rejected which entail the involution of the skin into the umbilical opening by means of buttons or conical pads. These merely enlarge the opening, and render the condition worse than it was previously. The usual method is to sew a flat disc, such as a penny or a piece of poroplastic felt, between two pieces of lint, to return the hernia and to strap the penny over the aperture. This, however, is not sufficient. Two folds of skin should be raised on either side, so as to overlap the disc, and then strapping should be placed crosswise over the opening. By this means pressure is taken off the weak spot, and cure occurs sooner. The strapping can be removed from time to time, but the attendant must be enjoined not to allow the hernia to come through, for the gain in improvement is rapidly lost, and the treatment must be begun over again. It should not, however, be forgotten that many cases of umbilical hernia become cured spontaneously as straining efforts are diminished, and as the general health increases. Very important is it to secure sufficient development of the abdominal muscles. In mild cases, and in those in which the hernia may be kept back by the pressure of a disc, exercises adapted to this purpose are of value. The child is directed to lie on the floor, and while the feet are held down, the child is made to rise to a sitting position with the back held straight. By this means the recti become well developed, the umbilical aperture is lessened, and the hernia gradually disappears. But it is evident that exercises are very deleterious in cases which cannot be controlled by a truss.

The question of operation may be entertained

in those cases in which the hernia is large, the muscles remain weakly, and the pressure of a truss fails to keep the hernia back. In such cases an incision should be made through the skin, the sac defined, the hernia reduced, and a purse-string suture should be placed around the margin of the umbilical ring after it has been refreshed.

Inguinal hernia.—Of far more importance than umbilical hernia is inguinal hernia in infancy. Unlike the former, which is more frequent in the female sex, inguinal hernia is found more often in boys and in the children of poor parents. But it is not seldom met with in the children of the moderately well-to-do, and even of rich folk. In either event it is a serious handicap to a child.

The forms met with are the truly congenital hernia, in which the funicular process remains patent throughout its entire length, and the intestine descends until it envelops the testicle. Secondly, the funicular hernia, in which the process is closed for some distance above the testis, but its upper part remains patent, and a hernia descends into it and forms a funicular hernia. Thirdly, the infantile form in which the same condition of the funicular process exists as in the second variety, but the hernia is pushed down behind it so that a cyst or rather a fluid-containing space exists in front of the hernia; fourthly, the ordinary acquired hernia of adults may also be seen in infancy. A fifth and very important variety is that in which hernia co-exists with an undescended testicle or a prolapsed ovary. Such a condition as the last named was described by me in the 'British Medical Journal,' October 9th, 1897. It was that of a child aged four months, who had on the left side a protrusion of bowel with interstitial inguinal hernia of both ovaries and Fallopian tubes.

The contents, however, of the sac in the hernia of childhood usually are intestinal rather than omental, since the omentum is so feebly developed that it can scarcely find its way outside the abdomen by these orifices. Not unusually, however, the cæcum and appendix may be found in it, and the latter may be recognised through the coverings. Such an instance I described as occurring on the left side in the article just mentioned. In this case the hernia became strangulated, and on cutting down on it I found on the *left* side first of all the appendix, and then

the cæcum. These were replaced, and the child made a good recovery.

Rotch records a hernia of the cæcum and appendix where an attack of appendicitis took place, which was operated upon with removal of the appendix, and a good recovery was made.

In infancy the anatomy of the inguinal region differs very considerably from that in adult life, and, indeed, strongly predisposes to the formation of a hernia.

Féré examined sixty-two children under a month old, and found that in only thirty-four was the funicular process completely closed on both sides. At first this process tends to close rapidly, but after the third week very slowly. Dr. Féllizet has pointed out that in infants of a few months old the external abdominal ring is relatively higher on the abdomen than in the case of young adults. Still further, the external ring remains patent over the whole length of the very short inguinal canal of infants. In the new-born child the internal abdominal ring lies almost immediately behind the external abdominal ring, and the length of the canal is often no more than the thickness of the abdominal wall. The direction of the canal is therefore from behind forwards; but as the pelvis develops, and the anterior superior spine and alæ of the ilia incline outwards, the canal is gradually lengthened until it attains its full size. The proportionate nearness in infancy of the spine of the pubes, Poupart's ligament, and the anterior superior spine of the ilium, explains the rarity of femoral hernia in childhood, since the femoral opening is small and well protected. If to the above conditions there be added weakness of the pillars of the external ring, or, as sometimes occurs, as is pointed out by Mr. D'Arcy Power, absence of one pillar, then we have all the factors present for the ready production of a hernia. It only needs, then, some slight extra pressure to force the bowel down, such as coughing induced by adenoids or by an elongated uvula or whooping-cough, straining as the result of constipation, calculus, worms, or an elongated prepuce.

With regard to the diagnosis of hernia in childhood, in a simple uncomplicated case it is easy, but there are certain points in which the affection differs in childhood from the adult. In a child it is often at a higher level on the abdominal wall. It also has less tendency to become scrotal, but above all it has a habit of disappearing from time to time.

Sometimes it may remain quiescent for as much as three days, and it is always unsafe, if the mother tells one the child has a hernia, to assert that no such thing is possible as the result of one examination only. But opportunity should be taken to examine the child from time to time, and at some more or less unexpected moment the hernia will be found down. It is also advisable to watch the inguinal canals during micturition, because there is no act which so readily brings down a hernia as this. As a rule rupture in childhood is readily reducible. But it may be impossible if the child is screaming; it is then not safe forcibly to reduce the swelling, but the child should be quieted or anæsthetised before any such attempt is made. Occasionally the rupture is irreducible; this may be due either to straining, in which case the difficulty is got over easily enough, as pointed out above, or the hernia may be obstructed by its contents or adhesions of the sac. Ashby and Wright mention of one of their cases that a large hernia was made irreducible by the presence of tubercular mesenteric glands, which had evidently enlarged after their descent, and it was only after removal of some of these and cutting of the rings that the rupture could be reduced. It is unusual for hernia to become strangulated in childhood. If it should be so, the greatest possible care should be taken in employing taxis, as damage may be readily done to the thin intestinal coats, and it is wiser, in place of attempting taxis, to operate at once for its reduction. The operation need not be at all extensive, since it is often sufficient merely to nick the pillars of the external abdominal ring, when the hernia will be found to glide back with great ease except in the case of cæcal hernia.

In the ordinary course of events, a swelling in the inguinal canal, resonant on percussion, opaque, with an impulse on coughing, can only be a hernia. But, as was pointed out some years ago by Mr. Howse, a descended coil of intestine full of flatus in an infant may be translucent. The conditions which may be confused with hernia are the following:—A hydrocele which may be either congenital, extending through the whole length of the funicular process and reducible from time to time into the abdomen; or a localised hydrocele which may be scrotal; or hydrocele of the cord. In either case the hydrocele is smooth, tense, and dull, while a rupture is necessarily irregular and resonant on

percussion. With regard to the congenital hydrocele, their reducibility differs very markedly. Some can be reduced with ease in the out-patient room, by placing the child on its back and gently raising the scrotum. Others are reduced only after a night's rest, and some form and disappear so slowly that it can only be supposed that a very minute canal forms a communication between the scrotum and the abdominal cavity, and the filling up of the hydrocele is often coincident with gastrointestinal disturbances, when it is quite probable that small collections of serous fluid occasionally appear in the peritoneal cavity. There may also occur fluid dilatation of the hernial sac, a condition which can only be verified by operation. With most of the preceding conditions hernia may be associated, and it is often difficult to distinguish them. If the hernia can be separated from the hydrocele the application of a truss may cure both. If not, it may be possible to withdraw the hydrocele fluid before putting on a truss. Lastly, the recognition of a small hernia may be difficult owing to the presence of a displaced testicle, but it is a cardinal rule in dealing with groin swellings of any kind to ascertain first of all that the two testicles are present in the scrotum. By so doing many mistakes are avoided. It has often happened that an undescended testicle the seat of inflammation has been mistaken for strangulated congenital hernia, but the mistake is readily avoided by exercising a little care. Sometimes a testicle will acquire an adhesion to the bowel and drag it down. The bearing of this upon treatment by trusses can be readily appreciated. A hernia can, as a rule, be diagnosed from a local abscess or a cyst by the absence of reducibility and the dulness of the latter. Similarly inflamed or enlarged lymphatic glands should give rise to no difficulty. Occasionally masses of fat surround the spermatic cord, and are nodular to the touch. These sometimes present difficulty.

The treatment of inguinal hernia resolves itself into three headings: 1st, the removal of all those causes which give rise to increased pressure in the abdominal cavity, and so force the bowel outwards; 2nd, treatment by mechanical means; and 3rd, operative treatment.

Under the first heading, removal of predisposing causes, the surgeon will examine the condition of the pharynx for adenoids, enlarged uvula or

pharyngitis, will see that cough is removed, will examine the prepuce, inquire as to any symptoms of calculus, ask if any worms are present, and examine the lower bowel for polypus. Having removed any such cause he can now begin to treat the hernia. In some cases in children, after the exciting cause has been removed, the hernia will get well spontaneously if the child is kept on its back, and the bowels are duly regulated. In other cases circumcision may be sufficient to prevent a return of the hernia. By way of keeping a hernia back in infants during the time they are wearing diapers, a skein of Berlin wool or lamb's-wool is employed at the Children's Hospitals. It is put on in the following manner. The uncut skein encircles the pelvis, one end being passed through the other at a point corresponding with the inguinal ring. The free end is carried between the thighs, and is fastened behind to that portion which encircles the pelvis. Such a one may be used during the day, and changed for a dry one at night. Particular care should be taken that where the knot presses, namely, over the inguinal canal, no excoriation occurs. This can be readily avoided by changing the skeins sufficiently often, and by use of zinc and starch or other drying powders. In children over three months I always advise the use of a properly made truss. The faults one usually finds with trusses for infantile hernia are that the pads are too small, and the spring is much stronger than necessary. Their use entails a good deal of trouble and care, and the following precautions should be observed. The skin should be washed once a day, and carefully dried, and some powder sprinkled on. If during the use of the truss any soreness occurs, the child should be sent to bed for a couple of days till it is healed. Two trusses, both covered with mackintosh, should always be at hand; one truss should be slightly larger than the other. When it is necessary to change a truss, the child should be laid down, the hand pressed under the truss firmly over the external ring, and kept there until the pad of the larger truss is in position. If the hernia is once allowed to come down, it will undo the work of the previous three or four months. All trusses for inguinal hernia in children should be double. In the first place, they stay in position better; and secondly, pressure over one inguinal ring only may give rise to rupture of the opposite ring.

A truss with an air-pad is often better borne by children than one with the ordinary horsehair pad. The pad of the truss should be nearly flat, and not convex, and peaked trusses are never required, the object of the truss being to prevent rupture entering the canal. From the pressure of ill-fitting trusses orchitis has occurred, and occasionally hydrocele, hence care is necessary in adapting them. It may be said that if, during the first year, a hernia is kept up by trusses without once coming down, the rupture will be permanently cured, but after the first year so favourable a result cannot be expected.

A congenital hernia can only be said to be permanently cured when the parts withstand some exceptional strain, such as the spasms of whooping-cough. But these congenital herniæ are apt to be deceptive. They will disappear for a long time, and suddenly reappear at a critical moment with such force and in such sizes as to present all the symptoms of strangulation. If a hernia is small, the rings well developed, and a truss faithfully applied day and night, a cure may result. But if after trial of one year the bowel still continues to present itself, or an undue impulse be felt at the inguinal rings, it is well to consider operation.

When to operate for congenital hernia.—(1) When the hernia is not kept up by the pressure of a truss. (2) When the parents are too poor to supply fresh trusses as the child grows. (3) When the pressure of a truss is badly borne. (4) If there be adhesion of the bowel and the testicle. (5) If there be an undescended testis, and pressure is badly borne, or it be difficult to fit a truss. (6) Generally in cases of cæcal hernia, since they are apt to become strangulated. (7) When the hernia is irreducible. The operation for the radical cure of congenital hernia is so safe that it should be more frequently employed. Indeed, the operation is often left till rather late. It is better, if the above indications are present, to perform the operation at an early age, and good results are obtained even in the youngest children. The method of operating for the radical cure of congenital hernia is not very different from that in adult hernia. The chief point is the method of dealing with the funicular process. Now the funicular process is always closely attached to the spermatic cord, and considerable difficulty often exists in separating them. The method I have adopted has been to lay open the funicular process at about its middle,

very carefully divide it transversely, then to separate it up with the utmost gentleness from the spermatic cord as far as the internal ring; here a ligature should be placed around it, and the excess should be removed. By carefully stitching together the upper margin of the lower part a proper tunica vaginalis can be made for the testicle. At the operation the wound should be firmly closed by sutures and covered with collodion and gauze, then with dressings, and finally with a piece of jaconet, and every care be taken that the child does not soak the dressings with urine. If an undescended testicle is present it may sometimes be replaced, but if difficulty occur it should be removed. The removal of such a testis is by no means a so serious proceeding as it may appear, since many of them are atrophic from the first.

Control of Nasal Hæmorrhage.—Dr. Gleason advises packing the nose with absorbent cotton, saturated with cosmoline or other bland oil. He suggests the following method for packing the nose, so that on its removal bleeding will not recur owing to the suddenness of removal. A strip of patent lint, eighteen inches long and one and a half inches wide, is saturated with cosmoline, folded near one end over a probe and pushed through the bleeding nostril into the pharynx, and the probe withdrawn. The nose and pharynx are now filled with a sort of bag, while the short and long ends of the lint proceed from the anterior nares. The long end is then folded near the ala nasi over the probe, and the loop carried into the bag within the nose and pharynx, which is gradually filled with loops of the strip of lint thrust in by the probe. The mucous membrane gradually swells, so that in twelve hours it is necessary to begin to gradually remove the packing. As soon as bleeding occurs, the detached piece is cut off and the remainder left undisturbed. Some hours later more is removed, and so in time all is taken out.

Laryngoscope, March, 1898.

WE have received from Messrs. Ferris and Company, of Bristol, a copy of 'Pocket Notes of the New British Pharmacopœia, 1898.' The notes are not intended to be an exhaustive synopsis of the 'British Pharmacopœia' issued early last May, but merely present in a convenient form information respecting the principal alterations and additions.

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* Specially reported for The Clinical Journal. Revised by the Author.

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DEMONSTRATION OF CASES AT CHARING CROSS HOSPITAL,

January 20th, 1898.

By STANLEY BOYD, M.B., B.S., F.R.C.S.

Cut Throat.

LADIES AND GENTLEMEN,—First of all I want to show you a case of incised wound of the throat treated by suture. Such wounds used not to be sewn up, lest air should be driven into the tissues, causing more or less wide-spread emphysema; also because such a wound would almost certainly have suppurated, and the pus, confined by stitches, would have burrowed extensively along the facial planes, perhaps into the anterior mediastinum. This woman had an incised wound of the neck, three inches long, and practically transverse, which passed through the thyro-hyoid membrane above the hyoid bone, and then through the pointed lower end of the epiglottis, by which it is attached to the back of the thyroid cartilage. There was a square opening one inch wide into the pharynx. The patient had bled a good deal, and was faint from that cause; otherwise she was in very fair condition. She showed the rapid pulse which these patients so frequently do present, and which is probably connected with their mental condition. We had no trouble in stopping the bleeding. I do not think I had to tie a single vessel, although the anterior jugular veins must have been cut across. Having cleaned up the surface round about the plugged wound with soap and water in the ordinary way, we turned our attention to the wound itself. We found that the knife which caused the wound had not been quite clean, as there was a black stain along the upper edge of the wound; therefore I cleaned the edges with a solution of 1 in 20 carbolic. The sewing-up then began. I attached the epiglottis to its base, and repaired the thyro-hyoid membrane with three or four stitches of fine catgut, passed with a small

curved needle, all being left loose until the last was in; then they were tightened. Through the wound, thus closed, it was very unlikely that air would be driven, and union by first intention was probable. Next came the muscular layer; I sought out the cut ends of the infra-hyoid muscles, and sewed them up with equal care with a continuous catgut stitch running across the whole of the wound. Lastly, the skin incision was similarly closed. The patient has worn no splint or cap to maintain flexion of the neck. I limited the movements only by means of the dressing, which forms a very excellent splint. It consists mainly of a long piece of gauze collodion on to the front of the neck from the chin to the sternum, applied whilst the chin is depressed. If she raises her chin she pulls up the skin below the wound. Over this some wool and a bandage were applied. It seemed to me that with this dressing we might do without the various contrivances which used to be employed to push the head forward and keep the edges of the wound free from tension. The chief reasons why we can now adopt this method of treatment by suture instead of leaving the wound open are, first of all, that we can disinfect the wound with fair prospect of success. Of course, if the disinfection had failed, or if, by carelessness in sewing up, I had infected the wound, suppuration would have obliged me to take the stitches out. The second reason is that we now stop bleeding carefully and sew up our wounds accurately. Formerly surgeons were really afraid to sew up very accurately in such a case; they did not expect union by first intention, and they knew well the dangers of pent-up pus. Now, in the belief that we shall not get suppuration, we sew up with every possible care and endeavour to bring all parts into their normal relation with one another; hoping thereby to prevent emphysema and late infection. You see that the result in this case is quite satisfactory; the wound, now a week old, has healed throughout.

Sigmoid Colotomy for Obstruction.

The next case is one of sigmoid colotomy in a woman of 30. The history is that, a year ago, she was in Waterloo Bridge Road Hospital for ovarian tumour, and you can see the small scar in the median line through which Dr. Gow took away the left ovary. Six months ago she began

to suffer from what she called inflammation of the bowels; but she does not seem to have had any symptoms of enteritis or of peritonitis—no pain, no distension, tenderness, vomiting, or diarrhoea; in fact, the difficulty was quite the other way—there was a difficulty in getting the bowels to act, or even in passing flatus. Sometimes the bowels were very much more troublesome than at other times, and when they troubled her most the abdomen became distended; then she would take purgatives, and the swelling would subside somewhat, but on the whole distension of the abdomen increased. The difficulty with the bowels and flatus became greater and greater, and colic more and more severe; she could not take food, lost flesh markedly, and had occasionally a little—but only a little—vomiting. Finally she was sent here to see Dr. Routh, who found a greatly distended abdomen, exposure of which caused the colon to stand out prominently with painful cramp; the front of the rectum was set in a tumour implicating the left broad ligament, which drove the bowel back against the sacro-iliac synchondrosis. There was no doubt that this mass was a recurrence in the left broad ligament of the tumour which had been removed by Dr. Gow on the occasion I have referred to. We have written to the Waterloo Bridge Road Hospital to try and learn the nature of the tumour, but have not yet had any reply. I should not be surprised to hear that it was an ordinary ovarian cyst, such as one assumes will give no further trouble after its removal. We are learning more and more that there is a great, though at present unrecognisable, difference between these tumours, in that certain of them recur and that the proportion which do so is not a very small one. We cannot tell even by microscopical examination whether an ovarian cystoma is going to recur. Quite lately I saw a case, at the New Hospital for Women, in which a woman aged 36 had a tumour in the right groin. It was very hard, and presented the outline which glandular tumours usually present, but it lay further in over the pubes than the glands generally lie. I felt doubtful as to the exact nature of this swelling; but the facts that it had nipped the skin, that it was very hard and tended to involve surrounding parts, made me certain that it should not be left where it was. I should say that the whole of the genital tract had

been examined, and the surgeon in charge was certain there was no primary malignant disease there. Well, the tumour was taken out. On section the mass was fibroid, lying in the fat which had been freely removed around it; in its meshes there were numerous small masses of colloid material. It presented altogether an appearance which I was not familiar with in that region.

Examination by the microscope revealed the structure of an ovarian adenoma. There were small round and irregular spaces lined by long columnar epithelium, with the colloid material in the centre. It seemed most likely that this growth in the groin was secondary to a columnar-celled primary growth as yet undiscovered. I suggested an ovarian tumour, but there was no evidence of one. There was a pelvic tumour, but it was regarded as certainly fibroid.

To return to our present patient. When she came in here she was very thin, as you see, and suffering much from colic; her abdomen was very greatly distended, but the laparotomy scar, though considerably widened and stretched, had not yielded. It was obvious that relief was urgently necessary, although there was no vomiting; so I did a sigmoid colotomy as soon as possible. It was not a very easy operation. There are two incisions which are used in colotomy ordinarily; one is made between the anterior superior spine and the umbilicus, and the other is made one and a half to two inches above the outer part of Poupart's ligament. I think the latter is preferable, because, as a rule, you can get the colon out through it more easily. I made an incision two and a half inches long, and had the very greatest difficulty in getting any part of the sigmoid flexure out; it was so distended that it seemed to fill the whole of the lower part of the abdomen, and so tense that it was almost impossible to drive the contents of a small segment either upwards or downwards, and to thus reduce its size sufficiently to allow it to be drawn through the wound. I did not want to make an unusually big opening in the abdominal wall for obvious reasons. However, after some trouble I managed to get out a coil, which I opened at once; and, taking every care to guard my wound, I liberated a lot of gas, and an enormous quantity of fæces escaped. As the bowel collapsed I was able to pull it further out, and allow the further escape of

its contents with greater safety as regards infection of the wound. I did not endanger the peritoneum with fæces in any way, but one edge of the skin wound was soiled and was carefully cleaned. Finally I got a complete loop of the sigmoid out. I then fixed the mesocolon with a stitch to the upper border of the wound. If you do not do so, no "spur" is formed; the way from the upper to the lower piece is quite easy, and the patient then has the inconvenience of an artificial anus in the groin, but still has to suffer the pain caused by some of the fæces passing the colotomy wound and on through the rectum. To avoid that you must get the bowel thoroughly kinked, so as to leave a "double-barrelled" opening. Such an opening we have here to perfection. There are two openings, which run one into the lower, the other into the upper part of the colon, and the mesocolon is attached to the skin between them. There is no chance of fæces passing from one into the other. It is perfectly easy to wash out the lower end of the bowel from this wound should mucus collect in it and decompose, giving rise to irritation.

To go back—I had got the bowel open, but I was nevertheless anxious to maintain the condition of asepsis. If I had simply fixed the bowel to the skin and peritoneum all round by stitches, and had left the fæces draining over the wound, I should have got a considerable amount of inflammation, though possibly not peritonitis; so I made use of this thick glass rectangular tube which I show you. It is very useful, and was suggested by Mr. Paul, of Liverpool. I had made the incision into the sigmoid about one inch long, and when the rush of fæces had ceased I carefully passed the flanged end of the tube through this opening and stretched the bowel over it. Then I drew the bowel forward, and with a piece of narrow tape I tied the bowel on to the flange, cleaned away any fæces, and iodoformed the junction well. To the free end of the glass tube a large rubber tube was slipped to carry the fæces as they escaped into a bottle or other receptacle fixed to the patient's side. An antiseptic dressing round the tube and bowel and round the wound was next applied, and a bandage was put on round all, so that all moved in one piece, including the receptacle for the fæces. At the end of three days ulceration will have taken place round near the tape, but in these three days deep union will certainly have occurred, and pos-

sibly union right up to the skin. We can thus keep a wound aseptic for the first three days—a most important point. This tube is capable of many other important applications.

The next question is how best to treat colotomy cases after opening the bowel, the chief object being to keep the parts free from irritation. If you take a piece of linen and rub boracic ointment or iodo-vaseline into it on both sides, so as to get it saturated, you will find the linen will adapt itself perfectly to the skin, so that there will be no gap underneath it. It does not of itself tend to absorb the discharges, and as you have oiled it so thoroughly, watery discharges will not mix with it. This you lay close round the bowel over a wide area of skin; then receive the faecal discharges in an oakum pad, making a little nest of oakum round about the bowel.

Aneurysm of the Common Femoral.

This case is the most important I have to show you to-day. The man's age is about 40; he is a bricklayer, but he was in the army from the age of 18, for twelve years in the line, and two years in the army medical corps. He left the service in 1886, and since that time he has worked as a bricklayer. He says he often used to push his shovel with the near side of his thigh. The case, indeed, was one of aneurysm of the femoral artery high up—a trouble which might have resulted from injury of the artery; but careful questioning showed that the point with which he used to press the shovel was four inches lower than the spot at which the aneurysm existed. There is no history of syphilis. He has always drunk heavily, and this is an important point, leading to frequent stimulation of the heart, with irregular increase of the blood-pressure, and the disastrous effect which alcohol has upon the vessels generally. He was first admitted under my care in November, 1895. He then stated that for three months he had noticed a swelling in the right groin; this measured about three inches vertically, and two inches in cross diameter. It lay immediately below Poupart's ligament, running down the course of the femoral artery. The right thigh as a whole appeared larger than the left, an impression which was borne out by actual measurement. The limb looked somewhat congested, and a good many veins were seen over the surface,

dilated, in the right leg as compared with the left. Evidently there was considerable pressure upon his femoral vein where the saphenous opens into it. The swelling I have mentioned was firm and pulsating, about the size of a Tangerine orange, and a small movable gland was felt over it. The integuments were normal. The pulsation was found to be expansile—that is to say, when we got our fingers on either side of the swelling they were distinctly pushed apart by something dilating between them. Next we pressed a finger on the external iliac, and stopped the flow through it, the swelling becoming smaller. When we took the thumb off it, the tumour sprang up in a beat or two to its original size. This is of considerable importance in making a diagnosis between an aneurysm or swelling in connection with an artery into which the blood is pumped, and a tumour which is merely lying on an artery—for the latter would not vary in size. The swelling could not be shifted in any way so as to destroy its pulsation. That, again, is a very important point in the diagnosis of aneurysm, from a tumour lying over an artery to which pulsation is communicated. If we can shift the tumour or lift it up from the artery, then the pulsation in the tumour ceases; but in an aneurysm such a thing is impossible. This patient complained of a good deal of pain down the inner side of the thigh to the knee—probably from pressure on the internal cutaneous. The pulsation in the limb beyond the aneurysm, in the anterior and posterior tibial, was very feeble and difficult to feel. The external iliacs and abdominal aorta appeared to be normal, and no diseased vessel was found in any other part, except in the left groin, where the pulsation along the left femoral seemed to be rather plainer than it should be. The heart was normal, and there was no evidence whatever of any mediastinal tumour. That is another very important point in cases of external aneurysm—to make sure that the heart and great vessels are normal. On 30th November I tied the external iliac by Cooper's method without any difficulty. I tied the vessel with kangaroo tendon, and closed the wound as for the radical cure of hernia. After this the circulation in the limb quickly became normal. The limb had been pale and cool for a short time, but by the time we had finished the other operations of the day it was found to

be quite warm again. Later it became hyperæmic; but this condition soon died away, and then everything seemed normal. Next day we found that there was distinct pulsation in the sac. The question arose, was it due to free anastomosis, or was it that the ligature had yielded through the knot coming undone? During the next few days the notes state that, on the whole, the pulsation got less, until on December 6th it was scarcely perceptible. On December 9th I found that the pulsation was increased, and on December 12th the sac was larger, though still smaller than before the operation. On December 18th the sac became as large as it was before the operation, and there was a well-marked systolic bruit; pulsation was stopped by deep pressure on the external iliac, and I came to the conclusion that in all probability the pulsation was not "recurrent," but that the ligature had slipped. I decided that the treatment which would have been right for "recurrent" pulsation, viz. to excise the whole sac, would not be best in this case; but that I ought to look and see whether or not the ligature had slipped. On December 21st, therefore, I had to expose the external iliac again, and it was a very much more difficult task on the second occasion. It was to me a very interesting matter, because I have done a very large number of operations for the radical cure of inguinal hernia, and here was an opportunity to see what had happened in a wound, very similar to that of a "radical cure," which I had made three weeks before. The condition revealed was most satisfactory. When I opened the wound I found a small collection of serum between the skin and the external oblique; then I came upon a layer of loose connective tissue, in which lay the knots of the silkworm gut, which were holding together the external oblique; they were not causing the least irritation; they were buried in the areolar tissue, and there was no gap to be seen in the external oblique to show where I made the cut. I had to remove my stitches one by one, and make a fresh cut into the external oblique. When I got through the latter muscle I had a difficulty in lifting it up from the muscle which was beneath it—the internal oblique,—because where I had separated the two there was a very thin, tough continuous layer of connective tissue binding the two together like a layer of glue. I had to dissect the external oblique up

with the scalpel. Then I cut my two deep stitches, drawing the internal oblique down to Poupart's ligament, and again I had to use the knife to separate these parts. From the point of view of radical cure of hernia the healing was most perfect and complete, and promised the maximum of resistance to any fresh protrusion. When I got down on to the external iliac I could find no trace of any kangaroo tendon; so clearly this clumsy ligature had become uncoiled at some period. It is difficult to say when this happened, because we have the note that from November 30th to December 6th the pulsation of the swelling diminished, and I do not see why it should have diminished if the knot had come undone at once. This time, however, I determined there should be no mistake about it, so I put two silk ligatures round the iliac artery and cut it between; after that we never had any trouble from pulsation in the sac or anything else; the pulsation stopped absolutely, and was never felt again. We had no difficulty with regard to the circulation in the limb. It became pale at first, but ten hours afterwards it was extremely hyperæmic, and by the next day the circulation had become normal. From that time onwards the limb as regards temperature, appearance, and circulation was normal, and the wound healed up as easily as did the first one, and the aneurysm shrank. When he left, on February 26th, he still had a lump rather smaller than a walnut in the region of the original sac.

Now he has come back again, having been away twenty-two months. I will ask you to notice that the knots of the fishing-gut stitches in the external oblique can be felt half an inch below the scar in the skin. Formerly, like others, I was often troubled by sinuses forming in scars over fishing-gut sutures, and refusing to heal until the stitch was removed. I came to the conclusion that these sinuses were not primarily of septic origin, but that they were due to the stiff ends of the ligature pressing and boring their way through the feeble tissue of the skin scar over them; the track then became septic and refused to heal. To prevent this annoying sequela, I have adopted the following technique. Every suture is knotted thrice, and the ends are then cut separately, as close as scissors will lie to the knot. Should a knot come undone the ends will be short and

curled—not rigid points directed towards the surface. Next, I always use a flap incision in the skin, so that the scar in the skin does not correspond to the scar in the deep parts; thus, in a radical cure, the line of fishing-gut stitches in the external oblique is covered by a layer of healthy skin, as you feel in the present case: ulceration through this is much less likely to occur than through recent scar tissue. Since I have employed this plan of working (two years or more) I have not had a single stitch come out, whereas I had many before.

You can feel that there is a small, firm, motionless mass which marks the site of the aneurysm; not a single vessel can be felt pulsating in the limb—not even the popliteal; but the popliteal cannot be felt on the other side. He says that whenever he gets up the limb swells and prevents him from working; it does not swell in hospital.

When he was in the hospital before, we noticed that he had too much pulsation in the left groin at a site corresponding to that of the aneurysm on the right side, and before he had left the hospital there was a distinct swelling here, which was about the size of the one he has now. He says that four months ago, when he made his only attempt to do some work, this got a good deal bigger and somewhat painful. He has now come to see if anything can be done for it. He has got a distinct swelling, measuring two inches from above down by one inch from side to side; there is a gland on the surface, and the swelling is absolutely stopped by pressure on the external iliac. The question is whether that needs treatment. I do not think the disease has made any progress in twenty-two months. I have some reason for thinking that this man prefers residence in hospital to the workhouse, and residence in the workhouse infirmary to earning his own living, and I think that in all probability he could do light work without injury. So I do not propose to do anything surgical at present. But supposing that treatment should become necessary, what form would be best? I might tie the iliac on this side also; but there is a very strong tendency nowadays to go back to one of the oldest forms of operation—complete excision of a sac. There is a good deal to be said in favour of it in some cases, particularly of popliteal aneurysm. If you tie the femoral above a popliteal aneurysm,

and the aneurysm is cured by your operation, the aneurysmal sac becomes thrombosed, and the clot extends into the popliteal artery and blocks it; therefore it is obvious that the tendency towards gangrene must be considerable, for a double obstruction—at the point of ligature and at the aneurysm—is introduced into the circulation of the limb. If you excise the sac of a popliteal aneurysm there is no question about the cure of it, because the aneurysm is gone; you have inserted one block in the arterial supply, but at the same time you have removed much obstruction to the circulation in the limb, because the sac presses both on the vein and upon the smaller vessels through which a collateral circulation should be established. Even in this case, in which the double obstruction of the ligature of the iliac is almost reduced to one, I think there is a good deal to be said in favour of this treatment now that we can control hæmorrhage so much better than our forefathers, and have the anti-septic methods which they had not.

Double Direct Inguinal Hernia.

This man is thirty-four years of age, and is a tailor by occupation. A year ago he noticed a hernia on the right side. He had a chronic cough, and attributed the hernia to this. Subsequently he noticed that a small swelling was forming also on the other side. The swellings were a little unusual in their appearance; they lay over the inner part of the inguinal canal. If the man raised his head from the bed they came straight out at once. After they had bobbed up in that fashion you could easily reduce them, and insert two or three fingers into the cavity of the abdomen through the hole in the abdominal wall left by them, and you could feel the rectus and the pubic spine internal to your finger. We had, therefore, to deal with a direct inguinal hernia.

Last Saturday I operated upon him, and I do not remember to have had quite such an experience as I had with this patient. On cutting down on to the external oblique I found the pillars of the ring were separated right up to the muscular tissue. I divided the inter-columnar fascia right up to the muscle, got down on to the internal oblique, defined and raised its lower border, divided the fascia transversalis, and demonstrated the epigastric vessels outside the sac.

As I drew the peritoneum to the near side something came into the wound which was reddish, and from the top of it a thick cord ran up; that was the bladder with the urachus, so I had a considerable piece of bladder in the wound. To tighten up the peritoneum satisfactorily I had to dissect the peritoneum off an inch square from the side of the bladder. I was then able to tie the peritoneum, and I kept the whole sac and pushed it up outside my ligature. It was dead, but it served as a pad to strengthen the part. I did the same thing on the left side. Here I did not see the hypogastric, but I did see the bladder. The superficial structures, including the transversalis fascia, were sewn up in layers as usual.

Flat-foot.

This boy does not look as if he had flat-foot a few weeks ago; he has now got a very good arch. He is fourteen years of age, and a year ago began to work in a grocer's shop and was kept standing for long hours. The arch of his foot gave way in consequence, and the right one, particularly, rotated outwards until it was quite hollow on the dorsum. Nothing has been cut. This is the first case in which I have really succeeded with wrenching. It is quite true that by wrenching you can often get an appearance of inversion, but it is no correction of flat-foot, because it is due to movement in the transverse tarsal articulation, whilst the deformity occurs chiefly in the astragalocalcaneal joint. He has been massaged, and has been encouraged to practise the exercise of walking about on the outer edges of his feet. He will have to relinquish his long standing, and in due time his feet will probably be quite strong.

Return of Testicular Sensibility in Tabes.

—Bitot and Sabrazes say Pitres has shown that analgesia of the testicles is present in about 75 per cent. of all tabetics. This symptom, however, is not so unchanging as some others, *e.g.* loss of the knee-jerk and the Argyll-Robertson pupils, and the authors record three cases in which the testicular analgesia, at one time present, later disappeared. In two of the cases, with the return of normal sensitiveness of the testicles there was also an improvement in the previously impaired sexual power.—*Medicine*, June, 1898.

A DEMONSTRATION OF CASES OF LARYNGEAL TUBERCULOSIS,

At the Hospital for Consumption and Diseases of the Chest, Brompton, March 9th, 1898, by

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LADIES AND GENTLEMEN,—I propose to give a demonstration to-day of a number of cases of laryngeal tuberculosis, beginning with those in which the vocal cords are affected. Then I shall show you cases suffering from a similar disease of the interarytænoid fold. Next I shall exhibit cases of tuberculosis of the epiglottis, and lastly, examples of disease of the greater part of the interior of the larynx.

Tuberculosis of the larynx for the most part is a disease secondary to disease of the lungs. It is the result of inoculation of the larynx by sputum coming from the lungs. The common situation of the process is the posterior part of the vocal cords, the interarytænoid fold, and the laryngeal surface of the arytænoid cartilage. Next in order comes the epiglottis, and last of all the ventricular bands. As to the reason why these particular parts are singled out there is some uncertainty. It has been suggested by Klebs and others that the vocal cords are affected because the sputum lodges in the ventricle, and so trickles over the surface of the vocal cords. That I think is not very probable, because the ventricles are lined by ciliated epithelium, which must keep up a vigorous movement; but it is quite possible that sputum may rest on the ledge which is formed by the upper surface of the vocal cord. However this may be, it is a fact that if you examine the larynx in foggy weather fog-stained sputum will mostly be found to adhere to the posterior wall, as if it travelled along the posterior surface of the larynx. Tubercular disease of the larynx, as of other mucous membranes, consists of two stages—first of all infiltration or deposit; secondly, ulceration. In many cases, although we see the patients at apparently an early stage of the disease, we find an ulcer without any evidence of infiltration. There has been some dispute whether,

in such circumstances, the disease goes through the first stage, namely, infiltration. I think there is no doubt that it begins as an infiltration. This is well seen at times in the case of the vocal cords, where the earliest recognisable change is a slight elevation of the surface due to a subepithelial growth, which subsequently undergoes ulceration and necrosis. From analogy we may fairly conclude that all tubercular processes in the larynx begin as an infiltration, and that ulceration is a secondary consequence.

I will now show you the cases, and make a few remarks upon each.

In the first patient, a man of 45, suffering from pulmonary tuberculosis, a slight hoarseness drew our attention to the larynx, and on examination we found the following condition: the right vocal cord was congested and thickened, and in its middle third there was a small nodular projection. Localised congestion of one cord is suggestive of a local lesion, and may be the first indication of tuberculosis or carcinoma. In this case there is a circumscribed infiltration of the cord, and we shall wait for the appearance of ulceration. If we find distinct ulceration we shall proceed to treat it with lactic acid.

The next patient, a man aged 22, the subject of pulmonary tuberculosis, was first under my care in December last. At that time he had ulceration of both vocal cords, and was treated with lactic acid. For a time the ulcers healed, but they broke out again, and the following condition was noted a fortnight ago: swelling and ulceration of the middle third of the right cord, and some swelling of the opposite ventricular band. In addition there was marked thickening of the arytaenoid fold,—that is to say, a prominence filling up the space between the posterior extremities of the two cords. After a second course of treatment with lactic acid there is still a little prominence of the right cord, but no ulceration. It is a question whether there is an ulcer on the opposite cord. I think that the slight depression of the surface which you can see represents a scar where an ulcer existed before. The disease of the vocal cords has been arrested, but there is still an infiltration of the interarytaenoid fold, in all probability a chronic ulcer. In chronic ulceration of the arytaenoid fold it is common to find a prominence like this. The edges of a tubercular ulcer are generally heaped up, and it is diffi-

cult to see the base. I will show you presently a patient who has a similar swelling, but it is not tubercular. As the process in the present patient seems to be arrested, we shall not apply any more lactic acid at present.

Next I have to show you a young woman. Six years ago she came to the hospital on account of hoarseness. She was rather anæmic at the time, and had lost some flesh, but although we examined the lungs carefully we found no physical signs of disease. The laryngoscope revealed diffuse ulceration of both vocal cords, with a crumbling, ragged surface and a thickened interarytaenoid fold. We examined the sputum very often, but only on one occasion could we find any tubercle bacilli. Repeated examination of the chest, extending over a period of five or six years, has given negative results. After numerous applications of lactic acid the ulceration gradually began to disappear. You will find now nothing more than a slight thickening and congestion of the vocal cords. There is still swelling of the interarytaenoid fold, probably due to a chronic ulcer. I believe this to be a laryngeal tuberculosis, which has been cured in one part but not in another.

The next patient is a contrast to the others. Two years ago we found a marked thickening of the interarytaenoid fold, the swelling consisting of two plate-like elevations separated by a shallow median fissure. I came to the conclusion that it was a case of pachydermia of the larynx, and as it was the year of the International Medical Congress I had the opportunity of showing her to some distinguished laryngologists, who agreed with the diagnosis. When I took her to the Laryngological Society some months later there was a difference of opinion. Some thought it was pachydermia, others did not hesitate to say that it was a tubercular ulcer. I think whatever it is, it is not tubercular. She is well nourished, and has no symptoms except slight hoarseness. We have never found any tubercle bacilli, or any signs of disease in the lungs. Lastly, the treatment has been very successful, the application of the galvano-cautery at two or three points to the interarytaenoid fold being followed by a notable diminution in the swelling. She has still some thickening of the fold, and at the posterior end of the right vocal cord there is a minute nodule, a so-called singer's nodule. This patient, however, uses her voice

very little. I touched this small fibroma of the vocal cord with the galvano-cautery once, and produced a certain amount of destruction of surface, but it has reappeared. Unless it increases in size I do not propose to deal with it, because minute growths are very difficult to grapple with. The thickening of the arytenoid fold is extremely like that of a tubercular ulcer. It is an unusual site for pachydermia, which usually affects the processus vocalis.

In the treatment of tubercular ulceration we begin with 50 per cent. lactic acid, and in most cases after a few sittings we employ the pure acid. You must be very careful to cocaineise the larynx thoroughly before you apply lactic acid; it is because this is not done that patients sometimes experience great pain. We are in the habit of cocaineising two or three times, and then rubbing in the acid well.

The remaining patients I have to show you present lesions of the epiglottis. In the first case the epiglottis is swollen, reddened, and misshapen, the result of diffuse tuberculous infiltration. This is a very different form of disease, and prognostically a very important one, being far less amenable to treatment. This patient is under my colleague, Dr. Maguire, who has kindly allowed me to show him.

The next patient, a young man the subject of pulmonary tuberculosis, has pain in the throat on swallowing, without any great hoarseness. If you examine his larynx you will find marked redness and swelling of the left half of the epiglottis, but no marked change in the vocal cords. You will notice on the tip of the epiglottis there is a white patch, which is the result of the lactic acid which we applied for the ulceration. A practical point to remember in the treatment by lactic acid is that as long as the slough remains on the surface it is of no use to apply more acid. The epiglottis is specially prone to take on diffuse infiltration, because of the large amount of loose submucous tissue in this part.

The next is a very important case. It shows the same changes in the epiglottis, and in addition a comparatively uncommon form of tuberculosis of the tonsil and soft palate. The patient complains of very great pain on swallowing. At first we found superficial irregular ulcers on the tonsils and

soft palate, with greyish granulation in the latter position. By degrees the uvula became involved, and is now much swollen and oedematous. We are attempting to check the progress of the ulceration of the palate and tonsil by means of lactic acid, but the chances of success are not very great.

The next case came to the out-patient department on account of marked dyspnoea and stridor, both inspiratory and expiratory, and some pain on swallowing. The voice was weak and slightly hoarse. On examining the larynx we found diffuse swelling of the epiglottis and aryteno-epiglottidean folds, entirely concealing the glottis. He was admitted to the hospital, and after a week's rest in bed the swelling of the epiglottis slightly subsided, showing that some of the swelling was inflammatory. One can now see the outlines of the glottis, which is greatly narrowed and bounded by irregular greyish-pink looking walls, which are probably ulcerated. There is an important clinical point about the case, namely, that the physical signs in the lungs are very slight. At first that might seem unusual, but, as a matter of fact, it is quite what one expects. In cases of marked laryngeal obstruction auscultation is much interfered with owing to the impeded entry of air into the lungs. One has to trust very much to percussion in these cases. Stenosis of this degree raises the question of tracheotomy. That is an operation we never perform if we can avoid it.

Some years ago in America and in Germany the heroic advice was given that whenever there was any conspicuous swelling of the larynx tracheotomy should be performed, almost as a routine practice, so as to give the organ physiological rest. The operation does do that; but when a patient wears a tracheotomy tube he finds great difficulty in expectorating, so that infective sputum accumulates in the air passages and tends to set up an acutely progressive form of pulmonary tuberculosis. Intubation is contra-indicated in a chronic case of this sort. I should say, as a general rule, that if the operation can possibly be avoided, it is better not to do tracheotomy. If a tube is once inserted it must, as a rule, always be worn. Among all the cases we have had in the hospital during the last ten or twelve years I do not think there have been more than six which have been tracheotomised.

The last case is an instructive one, as it was an

instance of mistaken diagnosis. He came here at the end of January complaining of pain on swallowing, and on examination we found complete destruction of the right half of the epiglottis, the left half being represented by an irregular stump with a pale yellow ulcerated surface. On the right side the ventricular band and vocal cord were swollen and fused together, and on this side there was almost complete loss of movement. The left vocal cord was healthy. From the very pale look of the ulceration, and from the pain which the patient suffered, one was inclined to think it was tubercular, but we put him provisionally on iodide of potassium and mercury, and the result was very remarkable. There has been rapid healing of the ulceration of the epiglottis. Since we have investigated the patient more closely we find evidence of interstitial keratitis, and of Hutchinson's teeth. In laryngoscopic work the differentiation of syphilis and tubercle is sometimes most difficult. In the first place one requires to know the habitat of the disease. Tuberculosis by preference attacks the posterior part of the larynx, syphilis the epiglottis. But there are exceptions to this rule. The syphilitic process is more acute, and more definite cicatrization ensues, even without specific treatment. You may get a certain amount of cicatrization in tubercular disease, but it is the exception. Again, the history sometimes helps one, and examination of the lungs or sputum may remove all doubt. There are other cases in which we have a combination of syphilis and tuberculosis; in fact, some authors think that if a man has syphilis he becomes thereby predisposed to tuberculosis. Therefore you will see that these cases are sometimes very difficult to diagnose. What contributed to the confusion in this patient was the fact that he had pain in the throat, whereas in syphilitic ulceration, as a rule, there is a remarkable absence of pain, even when the ulceration is extensive. Moreover, as I have said, the ulceration was of a pale yellowish hue.

As to the selection of cases for lactic acid treatment, the most favourable are those in which there is distinct loss of substance without much submucous infiltration. It is useless to apply the acid unless there is a breach of surface; otherwise it will not be absorbed, it will simply produce a local eschar. But where there is distinct loss of substance without too much infiltration you may get very good effects. I know a patient who was

treated in this way for a deep ulcer in the arytaenoid region eight years ago, and the resulting scar is permanent now, although the pulmonary disease has extended. Many slighter affections have been relieved completely by this method; but the acid must be vigorously applied, and you must take care to cocainize the parts thoroughly first. In exceptional cases, where there is a persistent nodular swelling of the epiglottis, I have found it a good plan to touch the nodule with the galvano-cautery and rub in lactic acid afterwards, so as to give the acid a chance of penetrating through the broken surface. But you have to select your cases with great care. It is not very safe to apply the cautery to the vocal cord, because you may accidentally touch the other cord with your cautery. On the epiglottis, however, you cannot do much harm, and if there is a lump there which you think is tubercular the procedure is quite justifiable.

In selecting cases for local treatment one has also to take into consideration the general condition of the patient.

It is undesirable to apply irritating substances to a patient who has marked pyrexia and advancing pulmonary disease. Unless the patient's physique is fairly good, and the disease is not actively progressive, it is best not to trouble him with local treatment of the kind I have described. Of course *palliative* local treatment is another matter. Many of these cases are relieved by insufflations of morphia and starch, or by cocaine lozenges or sprays. Another very good remedy which is much used here is the Vapor Chloroformi Co., a steam inhalation composed of a drachm of glycerine of carbolic acid and ten minims of chloroform to half a pint of boiling water.

KEIFFER suggests as the direct cause of the myxomatous degeneration of the chorionic villi an endothelial proliferation leading to a vascular occlusion in the chorion, and he suggests that this vascular lesion may be due to emmenagogic drugs, such as apiol, piscidia, and viburnum. He reports a case in which such drugs were employed with the subsequent expulsion of a hydatidiform mole. Jacobi, on the contrary, states that these emmenagogues are very freely used, and the relative rarity of cystic disease of the chorion is a strong argument against Keiffer's theory.—*Univ. Med. Mag.*

SHORT CLINICS IN THE WARDS AND POST-MORTEM ROOM.

BY

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Retrospect of a Case of Double Aortic and Mitral Valvular Disease with Adherent Pericardium.

The young patient R. H—, æt. 21 ('Med. Reg.' No. 136, 1897, and No. 302, 1898) long known to you in the wards as the case of "large heart with four murmurs, and an apex-beat in the seventh space," has now passed through that painful agony which is the lot of a section of the sufferers from aortic regurgitation—for, of his four valvular defects, this was the chief. That which death now reveals to you is no less instructive than the clinical events we have observed together in the wards.

THE TREATMENT.

The late treatment was a failure, and it must be a failure in all cases where progressive lesions tend to abolish function. Whilst striving to relieve him I was pointing out to you how little hope there was in the attempt. His distress and exhaustion were not a temporary aggravation, but the climax in a long history of disease. At that stage ordinary remedies were useless; strong measures only could have had any chance of staying the downward course; and among them I thought of venesection.

The reasons which weighed against a general bloodletting have been justified by the state in which we find the heart, and to this I shall presently refer. The alternative to venesection was to drain off the fluids as much as possible, and to endeavour to lessen in that way the load of the dilated heart. You have witnessed the failure of this attempt. The incisions of the ankle were of no avail, as in spite of the cardiac failure the œdema was too slight to provide any flow of serum. The method which you saw me adopt was to apply dry cups to the lower part of the leg, and to make

incisions, after antiseptic precautions, in the centre of the resulting swellings. This is a modification of the old wet cups. Dry cups had previously been applied to the back of the chest, but without any perceptible alleviation of the dyspnoea.

As to the previous treatment adopted during the last two months, the *rationale* of its variations may not have been very clear to you. It was essentially symptomatic; and symptomatic treatment cannot always be reasoned out. It is governed by opportunity.

Digitalis on the one hand, nitrite of amyl and tetra-nitrate of erythrol on the other hand, are almost opposed in their action. They were administered in succession, but for different purposes. As a fact the cardiac lesions in this case carried with them conflicting indications; and, again, the phases in the symptoms varied.

Cardiac sedatives such as nitrite of amyl and the tetra-nitrate are essentially symptomatic agents, meeting the emergency but not striking at the cause. Digitalis in its beneficial action is a constructive remedy, leaving something as a permanent result after its administration. In this case digitalis did no perceptible good except, perhaps, quite at the last, when it was subcutaneously injected with strychnine. The nitrite and the tetra-nitrate gave temporary relief from the angina, but did not stop its recurrence.

The only remedy which gave striking effects, and this was during an attack of pyrexia which I judged to be due to rheumatic endocarditis with slight shoulder pains, was salicylate of soda in combination with alkali. Whilst relieving the rheumatic pain, it eased the heart.

This attack was the last strain, after which the patient never recovered cardiac energy. The pain and dyspnoea, which soon returned with increasing urgency, ceased to be under the influence of all remedies, including inhalations, postural treatment, &c. The last complication was constant retching, and inability to retain even fluids, a result partly nervous, partly mechanical, and partly due to catarrh; and in this connection I pointed out, when the belly was opened, that the stomach was dilated as well as pushed down by the diaphragm. At this stage subcutaneous injections of digitalin, of strychnine, and of morphia were the only available treatment, and under their influence he gradually sank.

The Nauheim treatment.—The case is remarkable in connection with the great success which had previously attended this treatment after a long persistence of severe symptoms. When it was applied nearly eighteen months ago the patient had been repeatedly warded for considerable periods for cardiac dyspnoea and angina; and he had been practically unfit for work through breathlessness for a long time. I seized the opportunity of his being free from his most urgent symptoms after general treatment, to endeavour to reduce the size of his unusually large heart. Resistance movements and mild Nauheim baths were used. Their beneficial effect was surprising. After a course of five weeks he was able to leave the hospital; and he subsequently followed his occupation as light packer without interruption for eighteen months.

Thus angina, when due to this form of dilatation (which is quite distinct from that due to atheroma), and the existence of several valvular murmurs, need not be a bar to this treatment. At the same time we cannot be too careful in venturing to recommend it in advanced conditions. Each case must be decided on its own merits; but as a rule you should regard extreme cases of this kind as unsuitable for the Nauheim method.

Remarks on treatment.—The case is full of clinical teachings, and most of these have been conveyed in the history itself. The most important lesson is that, with the advantage of youth, we need not despair of some degree of recovery if a *restorative treatment* be adopted.

A purely symptomatic treatment is too often inefficient as regards the disease itself, and it may divert our thoughts from more thorough methods. There cannot be any doubt that the benefit previously obtained in this case was due to the restorative agency of the Nauheim treatment. The suitable combination of rest and exercise gradually raised the heart's energy to the level of its task. You will note that this is a question of nutrition. The more I observe the more do I feel that nutrition is the first essential for therapeutic success; and that we may look in vain for the specific actions of remedies if the functions of organs are starved. This conclusion is specially illustrated by the later history of the case, when drugs lost their effect because the heart's energy was worn out.

THE MORBID APPEARANCES.

(1) *The pericardial agglutination.*—On examining the organs the first thing that strikes us is the great size of the heart, and the space which it occupies in the left chest. Of all this we were aware.

It happened that this case had been used by me in the ward to demonstrate to one of my colleagues the fact that the dorsal spine is normally resonant on percussion; and so it proved to be in this subject, in spite of the unusual degree of *hypertrophy and dilatation* of the heart, and of the uncommonly low level of its apex. This is worth mentioning as a confirmation of my invariable experience to the effect that in the adult the *lower dorsal dull patch*, which always results from pericardial effusion, is never found in cases of enlargement of the heart without effusion.

But in addition to its enlargement, the heart was adherent to the diaphragm; and this had not been diagnosed. In agglutination to the diaphragm, closeness of the adhesions is the usual condition. Elsewhere the pericardium may develop two sets of adhesions, the *internal* and the *external*, each of which may be either close or loose, but at the floor of the pericardium the internal agglutination is the only one which can occur, and when complete it implies fixation of the inferior ventricular surfaces to the diaphragm. The *external* pericardial surface in this case was not tightly bound down to its surroundings, except to the sternum, where the loose areolar connective tissue had stiffened into fibrous tissue; but the *internal* adhesion was intimate and agglutinative throughout, or, as it is conveniently termed, "oblitative."

Laterally the external adhesions connecting the pleuro-pericardium and the lung had taken the shape of numerous fibrous cords of varying thickness and length. The length of these bands gives the range of movement between the two organs. Much of this mobility probably belonged to the pulmonary surface, but part of it we may assume to have belonged to the heart. It is the combination of internal and external agglutination which does most harm, inasmuch as it seriously hinders the heart's movements. Simple internal adhesion, when free from thickening, and when it does not implicate the diaphragm, interferes but little with the action of the heart. But a hide-bound con-

dition of the myocardium may be the result of internal agglutination alone when associated with thickening, and increased labour of systolic contraction as well as a corresponding impairment of the diastolic elastic recoil must follow. In this instance the external adhesions were not of the worst type, and some lateral excursion of the heart was permitted, although the inferior surfaces were immobilised.

(2) *The valves*.—Here again things were not quite as bad as they might have been. The aortic and the mitral valves are seen to be defective in both directions; but the changes are not extreme, neither is the functional defect.

The pure *chronic rheumatic* type of valvulitis is displayed in both valves. The aortic flaps are shortened and much thickened at their free edge, which forms a heavy, smooth rim of india-rubber-like toughness and pliability. But of atheroma there is no trace, either in the valves or in the aorta, which is perfectly elastic.

Similarly the mitral valve is thick and tough, but not atheromatous, and there is no absolute stiffening of the chordæ tendineæ, though they are thickened and shortened.

The remainder of pliability retained by the diseased valves enabled them to fulfil part of their office; and this explains the remarkable improvement which was maintained for so long a time, an improvement which would not have been possible had there been progressive stiffening and atheroma.

Nevertheless the structural consequences of the valvular defect were progressive, and particularly so the inevitable and fatal dilatation of the left ventricle.

(3) *The dilatation*.—Four dilating factors existed, but the three most important were the aortic regurgitation, the mitral stenosis, and the pericardial adhesion.

Great dilatation of the left ventricle with hypertrophy is, as you see, the leading change. The greater part of it may fairly be put down to aortic regurgitation; and I need not point out to you that its production is largely due to *loss of diastolic intra-coronary pressure*, a loss of pressure which must be very great whenever the sectional area of the leak is greater than the joint sectional areas of the coronary orifices. Hence the anginal attacks and the increasing enfeeblement of fibre,

in this instance evidenced by the striated aspect of the papillary muscles where fatty degeneration has set in.

At the same time adhesion to the pericardium cannot be left out as an additional cause of dilatation. And I regard the left ventricle as very prone to suffer early from the conjunction of internal and external pericardial agglutination.

Thus I have recently observed, at the two last obductions on cases of pericardial adhesion which I have witnessed, both being cases of children with completely adherent pericardium, and with early mitral stenosis, but without any aortic regurgitation, that the dilatation of the left ventricle was much more marked than that of the right. I believe that much of the right-sided dilatation in such cases is a later development, and that it is secondary in part to the increasing pulmonary difficulty due to direct pressure effects from the enlarged heart, and partly also to the early interference with the function of the left ventricle and of the mitral orifice, setting up mitral stenosis and incompetence.

These mitral defects I believe to be almost inevitable consequences, either late or early, of an obliterative internal and external pericarditis. In the present case mitral stenosis had led to the usual dilatation of the left auricle, but the right ventricle, though hypertrophied, was scarcely dilated. There was, however, some dilatation of the right auricle.

I had often speculated in connection with this patient's case as to the possibility of some mechanical relief to the circulation having perhaps resulted from a kind of mutual compensation between the several valvular defects; and the same question is still before us. This is too complicated a subject for discussion at this place; but it may afford you food for thought.

Erythema Exudativum Multiforme (Hebra).

—During recent years the idea has been gaining ground that the excitor of this affection is a bacterium in the blood. The efflorescences appearing on the back of the hands and arms are treated by the application of Liquor Burowi or lead water. Beside these applications, lead ointment is also used. The internal treatment, consists in the exhibition of sodium salicylate with antipyrin or phenacetin.—*Pediatrics*, June, 1898.

UTERINE TOILETTE.

By A. WARING, M.B.

THE cleansing and disinfection of the uterine cavity in cases of endometritis by such methods as lavage or injections, curetting, and applications of antiseptics or caustics to its interior are measures which, when efficiently performed in suitable cases, result in a marked improvement both of the uterine and constitutional condition of the patient.

Whilst, however, desiring to avoid unnecessary reference to the ordinary means employed with which you are familiar, my object in selecting the subject of Uterine Toilette has been to introduce the consideration of some details in the treatment by curetting, and more especially to review and discuss the methods and advantages of intra-uterine treatment in two varieties of cases.

Firstly.—In cases of endometritis associated with parametritis, perimetritis, or both; and

Secondly.—In cases of endometritis, where such conditions or complications as stenosis of the os uteri, acute flexions, versions, or misplacements, involve additional difficulties in the technique of the operation of curetting and subsequent treatment.

In order to appreciate the importance of rational principles of intra-uterine treatment in such cases of endometritis, I will briefly refer to some facts and matters of interest in relation thereto, before entering upon a discussion and reporting the results of treatment in cases which have come under my own observation.

The obscure pathological changes which occur in the endometrium and uterine tissue in endometritis, the difficulties of accurate diagnosis owing to the anatomical situation of the uterus, and character of the surrounding tissues and structures, the reflex local and constitutional disturbances characteristic of uterine disease, all these and many other circumstances lend special interest to the study and investigation of uterine disease and its treatment; but above all, a proper realisation of the significance of sepsis is essential to scientific principles of treatment.

Authorities have classified endometritis into cases due to organisms and those that are not, and

though endometritis may exist without the presence of pathogenic germs, yet the majority of cases which call for surgical interference in our practice are due to organisms—the only conceivable causes of a simple endometritis being due to such a congestion or hyperæmia from disturbed menstruation, &c.

Septic material once introduced into the uterine cavity may not only cause a septic endometritis, but may also result in the extension of infection to surrounding tissues and structures.

Invasion of the genital tract and peritoneum follows certain well-defined channels, and a clear understanding of these routes forms the basis of rational treatment.

Infection may spread from the uterine cavity by phlebitis, or thrombo-phlebitis producing septicæmia or pyæmia; it may spread by the lymphatics, producing cellulitis and peritonitis; or it may extend through the Fallopian tubes, producing a salpingitis, ovaritis, or peritonitis.

To elucidate still further the havoc which septic uterine contents may play in the production and extension of disease, it is necessary, for the purpose of realising the importance of surgical principles of treatment, to bear in mind that the os uteri, even in a parous uterus, is not sufficiently patent in most cases to allow efficient drainage, favouring thereby a retention or hindrance to expulsion of septic and inflammatory uterine contents.

The mucous membrane of the uterus, moreover, with its fragile surface, its crypts and glands, and its underlying abundant vascular and lymphatic structures, is readily susceptible to septic absorption, although by reason of this very structure it is, on the other hand, highly recuperative in its vitality.

Amongst organisms found in the endometrium and uterine discharge are—streptococci, gonococci, staphylococci, bacterium coli, combinations of cocci and bacilli, diplococci and amoebæ

The gonococcus, although classed amongst the most harmless, causes, in my experience, not infrequently a most severe and extensive peritonitis.

The virulent streptococcus is found in the endometrium in puerperal infection.

Evidence of infective invasion is proved by the presence of staphylococcus and streptococcus in tubal abscess.

We have, then, abundant evidence of the pre-

ponderance of sepsis in the production of endometritis, and secondarily also of the production of inflammatory conditions in the surrounding tissues and structures.

By such means as uterine injections, curetting, and applications of antiseptics and caustics, we are able in many cases to cleanse and disinfect the uterine cavity and remove the unhealthy endometrium; but these methods are sometimes unsuccessful, or do not suffice in effecting a complete cure.

One meets with a considerable number of cases who have been curetted, sometimes more than once, and derived no benefit.

Macnaughton Jones, in his recent edition of 'Diseases of Women,' makes the following statement:—"It is the experience of every surgeon that endometritis, both corporeal and cervical, yield occasionally to no treatment, or even when we have succeeded in altering the nature of the secretion, and have finally arrested it, a lull in the treatment is followed by a return of the old complaint in as aggravated a form as before."

This coincides with my experience, and in seeking for an explanation of this recurrence, clinical observation and the results of treatment indicate to me that amongst the most frequent causes of failure in restoring the endometrium to a healthy condition after curetting or other means, we must consider firstly the possibility of incomplete removal or destruction of the germ-laden, inflamed, or hypertrophied mucosa; and secondly, the inefficient dilation of the os uteri and consequent inefficient drainage, especially if acute flexion, versions, or other misplacements co-exist.

Otherwise failure can only be accounted for by constitutional or debilitated conditions, or by conditions which have been overlooked, such as malignant or tubercular disease, intra-mural fibroids, or disease of the appendages.

As an instance of a case in which the operation of curetting resulted in failure on two occasions, owing to incomplete removal or destruction of the diseased endometrium and inflammatory overgrowth, I will briefly give you a typical example.

The patient, a married woman of 34, was first seen by me on July 7th of last year (1897). She was suffering from severe menorrhagia and dysmenorrhœa, on account of which I was called to see her. The loss of blood was considerable, several

diapers being used daily. She was markedly anæmic and in a debilitated condition.

The patient dated the commencement of her illness from the time of a miscarriage two years previously; since then she had suffered from leucorrhœa, severe menorrhagia, and abdominal pains. Previously her health had been good.

About a month after the miscarriage she was advised to undergo the operation of curetting. This was done, and for a few weeks she was better, but soon the old symptoms recurred. The patient continued under treatment for nearly a year, and as she did not improve, she was advised to undergo a second curetting. This was done some four months before I saw her. No improvement followed this second curetting, and she was subsequently recommended to have her ovaries removed, to which, however, she did not consent. This is briefly the history that I obtained when called. On examining her I found the uterus movable, large, and tender; there was also some slight tenderness about both fornices, but no indication of gross tubal or ovarian disease.

I formed the opinion that the persistence of symptoms, seeing that curetting had failed to relieve, was probably due either to malignant or tubercular disease of the uterus or intra-mural fibroid.

She refused to entertain the suggestion of any serious operation, but consented to be placed under an anæsthetic with a view of examining and exploring the uterine cavity, of removing any fragment of growth for examination, and also with a view as a last resort of trying another curetting, and in addition, packing the uterus with gauze as described.

When under the anæsthetic I could feel nothing abnormal about the tubes or ovaries. Having fully dilated the cervix, I curetted the interior of the uterus with a sharp curette, and removed some fragments of tissue. The cavity was then swabbed with 1 in 20 carbolic solution, and packed fairly tightly with strips of iodoform gauze soaked in iodoform emulsion.

On the following day I removed the gauze from the interior of the uterus and re-packed it. This was done during three consecutive days.

To my satisfaction, the patient since the operation has markedly improved both in the local and general condition. She has menstruated regularly

during the last four months, without losing an excessive quantity and without abnormal pain, the most noticeable feature being the rapid and complete improvement of her general condition, which, as she states, is as good as it was before the miscarriage.

In this case, as in others, I believe that gauze-packing the uterus has a beneficial effect in promoting a healthy growth or cicatrisation of the curetted endometrium, as well as encouraging drainage of intra-uterine and peri-uterine inflammatory discharges or products.

Such an example as this must, however, not be looked upon as convincing evidence that it is frequent to meet with cases where failure after the operation of curetting is due to incomplete removal or destruction of diseased tissues, and if a second curetting has not succeeded it may be concluded that in the majority of cases we have most probably to deal with some condition which has been overlooked or unrecognised.

I have mentioned another cause of failure in the treatment of endometritis by curetting as inefficient dilatation of the os uteri, and consequent inefficient drainage, especially when acute flexions or misplacements co-exist with the uterine disease.

We meet with cases of endometritis where the cervical tissue forcibly resists free dilatation even by graduated metal bougies, so much so that we are only with difficulty able to dilate up to No. 10, giving just sufficient room for the admittance of a medium-sized curette. Such inefficient dilatation means inefficient drainage and possible retention of foreign material, and if an acute flexion also exists, much more so will this kinking tend to obstruct the outward and free flow of inflammatory or septic material from the interior of the uterine cavity.

I have noticed in several instances that this incomplete dilatation and subsequent rapid occlusion of the os uteri is followed by an elevation of temperature after curetting, that recovery is protracted, complete and rapid relief is not obtained, and that ill-defined uterine symptoms may continue.

Where there is any difficulty in obtaining efficient dilatation of the os uteri, I advocate packing with iodoform gauze soaked in iodoform emulsion as a simple and effective means of allowing a free discharge, and as a precaution against the possible

retention of foreign material. Gauze packing of the uterus and os, in several cases in which I have practised it, has been attended with most satisfactory results.

The gauze should be soaked in iodoform emulsion, so as to prevent it adhering to the raw surface. It may be removed and re-introduced without pain to the patient, and providing the necessary antiseptic precautions are taken, the question of risk need not be feared.

I shall now pass to the consideration of intra-uterine treatment in cases of endometritis associated with parametritis or perimetritis (pelvic cellulitis, peritonitis, and inflammatory conditions of the tubes and ovaries).

Hart and Barbour, in their recent edition of their work, make the following statement:—"There are truly inflammatory conditions of the uterine mucosa, and the importance of the endometrium as the starting-point of inflammatory conditions in and around the uterus has of recent years been emphasised. As the result of this, salpingitis and even peritonitis are regarded as secondary lesions, the important condition being that of the endometrium. This change of view in operative gynaecology is seen in three ways:

"(a) Intra-uterine treatment is being emphasised.

"(b) Pelvic inflammation is regarded as an indication rather than a contra-indication for curetting.

"(c) The more conservative treatment of the appendages is advocated."

In justification of the sound and rational views thus expressed, I shall further corroborate them by giving some examples of cases to which they are applicable.

It is reasonable to contend that where extra-uterine inflammatory conditions have originated from a primary septic endometritis, we should remove the focus of infection.

I have followed this principle of curetting, disinfecting, and, if necessary, draining by gauze-packing the uterine cavity, in cases of acute endometritis in which severe pelvic cellulitis and peritonitis existed, and obtained good results. In some of the cases the uterus and cervix were packed with iodoform gauze. This method of packing has been recommended by Polk, who states that in so doing drainage is ensured, and a secondary

influence is brought about on the surrounding inflamed structures, and that by an osmosis the inflammatory symptoms subside.

There are some who question the advisability of surgical interference in cases of acute endometritis associated with extra-uterine inflammatory conditions, and who treat them medically by rest, vaginal douches, external applications, &c. Patients so treated generally improve after passing through a trying and prolonged illness, but recovery is often incomplete.

The pyrexia, which may continue for a considerable period, results in anæmia and asthenia. The slow subsidence of the inflammatory process results in chronic thickenings, adhesions, flexions, neuralgias, and reflex nervous disturbances. It is therefore urgent that everything should be done to avoid such a result, and I am confident that by promptly cleansing and draining the uterine cavity, in spite of the co-existence of the most severe peri-uterine inflammatory conditions, we can prevent further destructive processes or suppuration taking place. We promote and encourage absorption of exudations, and effect thereby a rapid and permanent benefit.

The questionable point would seem to be as to whether one is justified in manipulating and interfering surgically with the uterus in the presence of inflammatory conditions in the surrounding tissues and structures. My opinion, based on the result of observation and treatment, clearly dictates that so long as ordinary precautions are taken to avoid rough handling, which might conceivably excite suppuration or cause hæmorrhage, we are justified in carrying out this rational treatment.

It is evident, however, that discrimination between cases is necessary; for example, while curetting might benefit a catarrhal salpingitis, the operative interference which it implies might be disastrous in a pyosalpinx.

I am able to record four cases of acute endometritis associated with severe peri-uterine inflammation which I treated during the acute stage by curetting, swabbing, and application of iodized phenol, and in two of which I also packed the uterus and cervix with iodoform gauze soaked in iodoform emulsion.

I will give a brief account of one of the most severe cases, which was probably due to gonorrhœal infection.

The patient, aged twenty-five, had been married two years. She had had no children, and there had been no miscarriages. When I first saw her in April, 1897, she complained of pain in the lower abdomen, nausea, and prostration. Her health previous to this attack had been very good.

The temperature was 102°6'. She told me she had had some vaginal discharge and painful micturition for about a week, but that this acute attack set in three days before I saw her with acute pelvic pain, headache, shiverings and nausea. She noticed some blood-stained discharge, but the catamenia had ceased a fortnight before.

On examination I found the uterus fixed, and there was a hard thickening of both fornices and pouch of Douglas. On abdominal palpation, resistant, hard, circumscribed areas of peritonitis could be felt extending in front to the level of the umbilicus, and somewhat higher laterally. There was no free vaginal discharge, but the examining finger was coated with blood-tinged mucus.

On my second visit the following day the patient was worse, all symptoms were aggravated, although the pain was relieved by hot vaginal douches, applications, &c. The temperature reached 103°4'. I resolved then to curette the uterus and drain it by packing with iodoform gauze, seeing that one had to deal with a non-parous uterus and a narrow os uteri.

This was done with the result that all symptoms rapidly subsided; three days after, the temperature became normal, the resistant areas of peritonitis rapidly disappeared, and in a fortnight she was allowed to get up, feeling well, and without any pelvic symptoms. It is now eight months since the operation, during which time the patient has enjoyed good health.

Not only acute, but also chronic peritonitis or cellulitis, which have been considered as a contra-indication to curetting, may, in certain cases, be regarded as a reason for removing the source of infection from the uterus which keeps up the inflamed condition.

Curetting and gauze packing the uterus so as to ensure drainage should be recognised as of value in its secondary influence on the Fallopian tubes in cases of salpingitis.

Without in any way depreciating the success or ease with which the uterine appendages can be removed by abdominal section, I agree with those

who state that the successful results obtained in abdominal operations has driven into unmerited neglect the more conservative and simple measures to which I have drawn attention.

In conclusion, I hope that my endeavour to emphasise the importance of intra-uterine treatment on rational surgical principles has not been altogether unavailing, and that a perfect Uterine Toilette should be considered not only as a means of restoring the uterus to a healthy condition, but also as an important preventative and conservative measure in inflammatory conditions of the surrounding tissues and structures.

[Delivered before the Nottingham Medico-Chirurgical Society.]

NOTES, ETC.

A Case of Epileptic Icterus.—In view of the doubt which a number of writers have expressed regarding the existence of epileptic icterus, Dr. Féré has felt justified in reporting a case which came under his observation in the early part of 1897. The influence of the central nervous system upon the biliary secretion has for some time been recognised in the pathological effects of emotions, and the existence of emotional icterus is no longer doubted. Icterus may be caused by an intense emotion, such as fright or anger, and may be developed immediately, as in a case reported by Bouilland, where the patient, who had received a disagreeable letter, was himself able to observe the phenomenon while shaving himself. In another case Diderot, who one day had witnessed an execution, came home with a very pronounced icterus. Sometimes the icterus does not develop for some hours, and occasionally not for some days. The long intervals have been observed in cases of long-continued worry. The emotional icterus varies in intensity and duration, but is, as a rule, curable, and is usually only seen in neuropaths. In an hysterical woman, thirty years of age, the emotion was caused by an attack of asthma in one of her children. It is also claimed that mental impressions of a melancholy nature, especially when they have been present for some time, may favour the formation of gall-stones, either directly through the resulting depression of the nutrition of the body,

or secondarily by causing a morbidly increased cerebral activity, and thereby an increased production of cholesterin. It has, furthermore, been maintained that gall-stones are more frequently met with among the insane than among the sane, living under the same circumstances. Cases have been reported in which icterus was reproduced every time there was a repetition of the same mental excitement. Thus Rayer speaks of a young man who became icteric every time he was angered. The physical condition present in anger is not without a certain analogy to the epileptic explosions; one may therefore expect to find icterus figuring as a complication of epilepsy. Even Hippocrates speaks of bile as the cause of epilepsy. Esquirol seems to consider icterus a common symptom in epilepsy; still Féré has only been able to find very few accurate reports concerning this in the literature, and he is therefore of the opinion that it is a very rarely observed phenomenon, *i. e.* if one does not include the yellowish pigmentation of the sclera observed before or after the attack in epileptics suffering from gastric disturbances. The case reported by Féré is the following:—A lady forty-nine years of age, of nervous disposition, but who had never before been subject to epileptic attacks, was two years previously, during her menstrual period, on an excursion, when a storm suddenly came up, and she was drenched by the rain. She was at the same time much worried about her daughter, whose health was likewise exposed. Her menstruation immediately ceased, and did not again return. Fourteen days later, while sitting at the supper-table, she was seized with the first epileptic attack. She became suddenly pale, fell backwards, began to have spasms, which were followed by deep coma and stertorous breathing. Her sleep continued until next morning, when she voided a large quantity of urine of a very dark brownish colour. She had not passed urine during the attack. A short time afterwards it was observed that the conjunctivæ were yellow, and that the alæ of the nose, the temple, and forehead were of a light yellow colour, which became more intense on the following day, and extended to all parts of the body. The urine became still darker, and the *feces* of a *café-au-lait* colour. On the third day the urine was not so dark, the *feces* less pale. The skin remained icteric for about eight days, and the

patient experienced an intense itching, and had a very slow pulse (second day, 28). During the following three months the attacks recurred every two weeks, being nocturnal as well as diurnal, the former occurring after a couple of hours' sleep. Each attack continued to be attended by a more or less intense icterus, and the first urine voided after each attack was of the same character as on the first occasion. Of late bromide treatment has been instituted, which has prolonged the intervals between attacks, but has not diminished their severity. In every case the icterus was reproduced. Between attacks the patient has not presented the least symptom of hepatic disease, nor does she suffer from any marked gastric disturbance. In this case there can be no doubt about the existence of a relationship between the icterus and the epileptic attack. With regard to an explanation of the phenomenon, Féré refers to the theory of Potain concerning emotional icterus. The moral check causes a dilatation of the abdominal blood-vessels, leading to a diminution in blood-pressure, thereby producing a condition allowing the bile to pass from the biliary duct into the blood-vessels by osmosis, &c.—*Progrès Médical. Hôpitalstidende*, November 3rd, 1897.

Foreign Body in Vitreous removed by Haab's Electro-Magnet.—Dr. A. Barkan has exhibited a patient, a very gratifying illustration of the splendid work done by Prof. Haab's large electro-magnet in removing a metallic body from the vitreous of the eye. He said, "The patient was struck in his left eye by a metallic chip, which, entering through a very small opening in the corneo-sclerotic margin, passed around the lens into the vitreous. The initial opening being very small, I preferred to get the foreign body by exposing it to the magnet in the anterior chamber. This was speedily accomplished, the patient experiencing pain for only a second. In the anterior chamber, though, the foreign body became entangled in the iris, and it was necessary to tap the chamber and cut out a small fold of iris, whereupon the foreign body was speedily attracted by the magnet and adhered to its pole. The foreign body was $\frac{1}{100}$ of a grain in weight; had an ugly hook-like shape. The wound healed rapidly, and patient recovered sight almost to normal extent. He reads smallest print, sees well at a distance, and

under ophthalmoscopic examination—which is a very remarkable point—the lens is seen to be entirely clear, the foreign body having taken its way out around instead of through the lens. This corroborates Prof. Haab's statement that foreign bodies from the vitreous attracted by the magnet into the anterior chamber will always go around the lens instead of through that body. This case is one of five which have been successfully removed by Haab's magnet within a little over a year.—*Occidental Medical Times*, April, 1898.

The Treatment of Dyspepsia.—In 'Revue de Thérapeutique' of Jan. 15th, 1898, we are told that Robin recommends the following prescription in those cases associated with pain and pyrosis:

℞ Hydrated magnesia	...	20 grs.
Subnitrate of bismuth	...	6 "
Prepared chalk	...	8 "
Bicarbonate of sodium	...	15 "

These parts are made into one powder, and twenty similar powders are given to the patient. One of these is taken three hours after meals. Should the painful symptoms be excessively severe, one sixtieth of a grain of hydrochlorate of morphine, or one sixth of a grain of powdered opium, may be placed in each cachet. For the arrest of acetic fermentation absolute avoidance of alcoholic drinks must be insisted upon, and where lactic fermentation is marked the quantity of fruit eaten should be limited. Such cases may be benefited by the administration of fluoride of ammonium 15 grains, distilled water 10 ounces, and one or two teaspoonfuls of this solution given three times a day when the discomfort is felt. Where butyric acid fermentation is present, which is a more rare condition, Robin uses the double iodide of bismuth, and cinchonidin under the name of erythrol, as follows:

℞ Erythrol	...	1-6 to 1 gr.
Hydrate of magnesia	...	2 to 4 grs.

To be placed in a cachet and taken after meals.

In many instances two small doses of arsenic or bromide of potassium or Fowler's or Pearson's solution may be given with advantage. In those cases where the dyspepsia depends upon hepatic congestion and cirrhosis, hydrotherapeutic measures may be resorted to, and mild alkaline waters, such as those of Vichy or Carlsbad, may be taken. In other instances the waters of Homburg are ad-

vantageous. In those cases where there is cardiac disturbance along with the dyspepsia the use of tobacco must be stopped, alcohol used in moderation, and tea and coffee taken only in small quantities. The evening meal should consist entirely of vegetable material, and bread should be forbidden. In the way of medication it will be useful to apply a certain amount of friction over the præcordial region, and it may be necessary where there is cardiac pain to apply the following ointment to the præcordium :

℞ Veratrin	2 grs.
Extract of opium	12 „
Essence of turpentine	30 min.
Essence of peppermint	12 drops.
Benzoated lard	1 oz.

A small part of this is to be rubbed into the præcordium once or twice a day. In other instances a capsule of ether will exercise a favourable influence in bringing up wind. Should there be marked dyspnoea along with cardiac disease, good results often follow the inhalation of oxygen. The gas should be slowly taken to the extent of five to ten quarts.

Should syncopal attacks come on, inhalations of nitrite of amyl are useful, and caffeine may be administered at the same time. For allaying nervousness, valerianate of ammonium may be given, or the following prescription may be used :

℞ Arsenite of potassium	1½ drs.
Cherry laurel water	2½ „
Ether	1 oz.
Essence of valerian	4 „

Sig. : a dessert-spoonful to a table-spoonful every two hours until the symptoms disappear.

Should the symptoms persist in spite of treatment, it will be necessary for the patient to leave his business, and go to a health resort for the improvement of his general condition.

Therapeutic Gazette.

Barlow's Disease.—Zuppinger reviews the literature and reports in detail the case of a 3¼-year-old girl, rachitic, with large swellings over both lower tibial epiphyses, subcutaneous hæmorrhages, and swollen, softened gums over carious teeth ; both forearms became swollen, and there was marked œdema. In four months the child was discharged cured, and the deformities disappeared.

Amer. Journ. of Obstet., June, 1898.

On the Nature of Rickets.—Chaumier ('Centralb. f. Kinderheilk.', 1897, ii, 358), from his studies, is of the opinion that rickets is an infectious disease, which may be contagious. This opinion is based on the frequent presence of rickets in institutions for children, and also on the fact that in case a child suffers from rickets in a house occupied by other tenants the rickets will also make its appearance in other children. He further observed that wet-nurses, whose own children were rickety, communicated the disease to the children they nursed. Casati has mentioned that he knows of families whose children born in certain houses were rickety, but those raised in another house remained free from the disease. The author became aware of epidemics among young pigs, in whose bones he later found the characteristics of rickets. He arrives at the following conclusions :

1. Rickets is a specific disease, caused by unknown microbes.
2. It is contagious, endemic in cities, and occasionally epidemic.
3. It is found in an epidemic form in young pigs, and its germs are preserved in houses.

Pediatrics, June, 1898.

By the Rectal Administration of Large Doses of Sodium Salicylate in Articular Affections, the desired effects are obtained promptly according to Harlet in the 'Nord Méd.' of May 15. He commences with 8 to 12 grams a day in two injections, each in a cup of bran water, adding a little opium or laudanum if necessary. It is completely absorbed in six to eight hours. He then decreases the amount by a gram every second day according to results. After seven grams, but one injection a day is given. He applies locally a 10 per cent. salicylic acid salve, to which a little turpentine or phenic acid has been added, and keeps the patient in bed ten to fifteen days after all pains and swelling have subsided, to prevent relapses or reinfections. This treatment puts an end to the pain in less than three days ; the affection leaves no sequelæ, locally or at a distance, and cardiac or gastric complications are avoided. He has been equally successful with it in acute or chronic articular rheumatism or hydroarthrosis, iritis, gonorrhœal joint affections, cold pleurisy, etc.—*Journ. Amer. Med. Assoc.*

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ON PRACTICAL POINTS IN PERCUSSION AND AUSCULTATION, AND IN THE PHYSICAL EXAMINATION OF THE CHEST.

ABSTRACT OF LECTURES TO ADVANCED CLINICAL STUDENTS

BY

WILLIAM EWART, M.D., F.R.C.P.,

Physician to and Joint Lecturer in Medicine at St. George's Hospital; Physician to the Belgrave Hospital for Children; Examiner in Medicine at the Conjoint Board.

I.

ON PLEXIMETRY AND ON PLEXIMETRIC BONES AND VISCERA.

THE subjects for these lectures have formed part of your earliest clinical training, and to those of longest experience among you they may perhaps sound like a tale twice told. You can all look back upon many teachings and upon an endless reading of books. Nevertheless much that I have to tell you is not to be found in the latter. The subjects to which I mean to refer are matters of practice, and they specially concern you as budding practitioners in view of your coming function of independent diagnosis. Having learned the approved methods and trudged through the monotony of routine, it is time that you should step more freely. We must now let you into some of the secrets, and I venture to hope that this initiation will mark for each of you the passage from the stage of tyro to that of the expert.

Being practical, much that I have to convey lends itself imperfectly to verbal description, and will be demonstrated to you on the living subject at ensuing meetings. The indispensable introduction to these demonstrations which must claim our attention to-day happens to be the most theoretic-

cal portion of the whole subject. But as we proceed you will perceive that it is less abstruse than the title might imply, and you will live to regard an acquaintance with pleximetry, and with all that which I have ventured to include under the heading of "Pleximetric Bones and Viscera," as among your most serviceable acquirements.

Pleximetry and the Pleximeter.

The pleximeter is to be avoided by the *beginner*. He should learn to use his fingers, and train their tactile appreciation by unaided percussion. This temporary necessity is often used as an argument—but it is a negative one—against pleximetry; it really does not touch the question as to the merits of the latter.

In your case, after acquiring sufficient dexterity, it is legitimate to avail yourselves of this simple aid. *The percussing hammer* is an unnecessary complication, and a possible source of error. The right hand is admirably suited for this purpose. Its only fault is the power which it possesses of unconsciously "manipulating" the sound by its fine range of velocities and weights and by its register of after-pressures.

Dr. Kingscote* has, however, lately re-introduced the percussing hammer in connection with his ingenious pleximeter, which does not work without it.

What is a pleximeter? Essentially any solid interposed between the percussing stroke and the surface to be set into vibrations. All solids are conductors of vibrations, but the selection of the *material* is important. There are two other points in which a pleximeter may be good, bad, or indifferent, viz. the size and the shape.

Too great a size has been the chief fault of many pleximeters. They should not exceed the dimensions of the finger, lest it should be impossible to use them for the percussion of small hollows. In the facility for doing this no instrument can beat the finger. Dr. Sansom's pleximeter, which has been found so useful that I have not wished for a better one, carries out this indication and provides us with two flanges both of the same width (one third of an inch), but of different lengths.

The shape of Piorry's flat ivory pleximeter is almost more convenient for the pocket than for

the work to be performed. For finer percussion it had to be used on its edge, and this was not satisfactory. The shape of Sansom's pleximeter enables

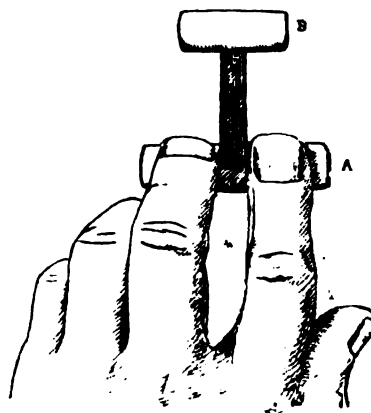


Fig. 1.—Sansom's pleximeter in use. For the percussion of small areas the pleximeter is reversed, and the small flange is applied to the chest.

it to be held between the sides of the fingers whilst the pulp will receive from the upper surface of the flange the tactile vibrations.

The faults and the advantages of the pleximeter.—A pleximeter may be of the wrong material, size, or shape. This alone would not constitute an argument against pleximetry. But there are inherent faults to every pleximeter.

(1) All solids yield, when percussed, a sound of their own varying, according to their density. This may be very slight, as in the case of soft india rubber or of any soft material; but vibrations would be very imperfectly conducted by them. We have to use, for the sake of conduction, dense substances, such as wood or vulcanite, and the sound obtained on striking these is of that high pitch which is the chief factor of the so-called "dulness" of percussion. It is obvious that when you strike the chest through a pleximeter this high-pitched note will add itself to the vibrations elicited, and that the percussion sound will be modified. Thus the percussion of the same spot will yield a different quality of sound according as you use the finger or the pleximeter.

This fault is really an advantage; and it is upon this peculiarity that the value of pleximetry is based. In a small pleximeter such as this, the superadded note of dulness is not considerable, but its presence causes any slight dulness to become perceptibly duller when percussed through it. In this way

* Cf. 'Lancet,' December 19th, 1897.

shades of dulness not otherwise readily perceived become obvious, and it becomes possible to trace out the dulling influence of a solid organ to the extreme limits of its extension. This determination of the actual size of organs can be carried out with rapidity and with great accuracy. The percussor acquires a well-justified confidence in his results; and in the hurried work of a busy practice he can get at his facts without delay and perplexity which any doubtful experiment entails. The pleximeter, in a word, is a fine *analyser for dulness*.

If, on the other hand, the part percussed should be resonant, the slight *twang* of dulness special to the pleximeter is lost in the low-pitched deeper and fuller thoracic vibrations, and no element of mistake is introduced.

The advantages and the shortcomings of the finger.—As regards the quality of the material the finger is unsurpassed. It is flesh and bone like the parts percussed, and like them it has very little percussion sound of its own. Provided it be pressed sufficiently firmly, it complicates matters only by adding to the thickness of the tissues which the vibrations have to traverse. On the other hand, like other soft substances, unless it be condensed by firm pressure, it does not favour the transmission of vibrations; on the contrary, it deadens them, and this is the secret of the great difference in the amount of sound obtained from the percussion of the same surface by different percussors. In this respect the finger is inferior to the pleximeter. I need not refer to the damage or pain which it may suffer from being too constantly used. One great advantage which it certainly possesses is the information which it derives from a direct palpation of the surface.

The relative value of the methods.—We owe to pleximetry the chief advances in percussion. Piorry's work in tracing out the dulness of organs was done with its aid. Skilful finger percussion claims to be able to equal the results obtained by the pleximeter; but it seems to have lacked that clearness and precision which enable us to discover new facts, and to demonstrate them to others. Indeed, the exclusive use of unaided finger percussion must be held responsible for sundry imperfect notions which have prevailed until quite recently, *e.g.* that the cardiac dulness and that of the liver could not be distinguished from each other, that the full extent of the anterior

cardiac surface could not be traced, and that its margin could not be defined with absolute accuracy; and owing to the same cause the dorsal dulnesses have been overlooked or ignored. Yet all these points may be easily made out, even with finger percussion, now that pleximetry has shown the way and supplied the demonstration.

The Pleximetric Bones.

All the component parts of the bony thorax are natural pleximeters for the underlying organs, and among them the ribs, although these, except for the finest work in percussion, are commonly disregarded. But the pleximetric function of the other bones is of practical importance, and a knowledge of it is indispensable. I refer specially to the sternum, the clavicle, the spine, and the scapula. Each of them tends to yield over its whole extent a uniform note, either dull or resonant. That is the general principle. But some of them transmit, in addition to this, local vibrations, so that the general note may be modified at various parts.

The sternum is the type of a pleximetric bone; and the resulting peculiarities of sternal percussion are generally put before the student. In most chests the sternum is resonant from end to end; but the expert will easily demonstrate that it is not evenly resonant. The resonance of the lower part of the sternum, which is so marked in spite of the solidity of the underlying heart, is purely pleximetric. This explains why the absolute cardiac dulness should invariably begin along the left sternal edge. Only when, owing to great cardiac enlargement or to effusion of fluid, the pericardial contents drive the right pulmonary fringe out of all contact with the right edge of the sternum, does the dulling influence assert itself. In its upper part the sternum is exceedingly resonant owing to the underlying trachea and pulmonary fringes. But below the manubrium, and above the level of the heart, there is a modification of this resonance—a slight partial dulness, which is often termed “retro-sternal,” and to which I have referred as the “pre-vascular dulness” in contrast to the precordial dulness because it is due to the great vessels.

The scapula is another instance of pleximetric resonance, or resonance by conduction; but its peculiarities in that respect have not received the same attention as those of the sternum.

The scapula is a huge pleximeter, and its per-

cussion draws sound from a considerable pulmonary area. This explains the remarkable fact that so large a mass of muscle and bone should be resonant instead of dull (see note (a), p. 226), its considerable resonance is rather a bar to fine localisations. Nevertheless, just as in the case of the clavicle, attentive percussion may obtain even small dullnesses which may be limited to portions of the pleximetric surface. And I would remind you that laterally and below it is possible by shifting the patient's shoulder, or by raising the arm, to get at the thoracic surface itself, and this should be done in doubtful cases.

The pleximetric character of the scapular resonance is easily proved by comparative percussion, *i. e.* by percussing alternately the surface of the bone and the thoracic surface all round its border; and, again, by causing the scapula to shift we can percuss one and the same spot (say in the interscapular region) alternately when that spot is uncovered and when it is covered by the shoulder-blade. In health the percussion will be markedly more resonant (which is an apparent paradox) when the scapular mass intervenes and increases the thickness of the thoracic wall.

On the other hand, in given conditions the scapula may, likewise after the fashion of any pleximeter, add an increased dullness to the underlying dull area.

The mobility of the scapula and its size render it a striking and demonstrative instance of the pleximetric character of bones. But there are others.

The clavicle is strikingly pleximetric, *i. e.* capable of conducting vibrations. A glance at the skeleton will enable you to realise this fact. You will observe how small is the upper aperture of the thorax, and therefore how small is the surface of pulmonary tissue which the clavicle overlies. Yet this is enough to render the entire length of the bone resonant on percussion.

Although the left apex is slightly narrower than the right, and in spite of other slight anatomical differences, the two clavicles yield in health percussion notes which are practically speaking identical. This is an important fact for the purposes of diagnosis.

I have implied that the clavicle is resonant from the sternum to the acromion, although it spans areas possessing the most different percussion

values, between the highly resonant tracheal region and the exclusively solid masses at the tip of the shoulder. This is another important point of great value in diagnosis; but I shall call your attention in my next lecture to the fact that the clavicular resonance is not of the same degree throughout the length of the bone, but that under the influence of the surroundings it varies from point to point.

Fig. 2.

From a photograph taken by Mr. H. G. Drake-Brockman.

The spine and spinal percussion.—The spine is another natural pleximeter. Percussive exploration of the spine does not seem to have been practised; yet it is of great diagnostic value in many cases owing to the definiteness of its results. If you should percuss a healthy spine—and the best way to do this is to apply the finger or pleximeter to the spinous processes, direct percussion being somewhat painful to the patient—the note will be resonant all the way down. This is easily explained by the proximity of the larynx and trachea to the cervical spine, and by the close contact between the dorsal spine and the lungs. Importance attaches to this invariable resonance,

which gives great significance to the occurrence of any dulness. Where any source of dulness exists to the right or to the left of the spine, still the spine remains resonant by reason of its contact with the other lung. Often when a moderate dulness prevails on both sides of it, nevertheless its resonance persists.

There is another fact of great practical importance. When, owing to some local pathological condition the spine is rendered dull, the dulness remains limited to that region affected by the local condition.

In addition to the resonance special to the spinous processes, there is also on either side of the middle line a narrow strip of resonance corresponding to the width of the vertebræ, and this may resist some of the slighter dulling influences.

This pervading resonance of the spine and vertebræ is one of the factors concerned in the production of the curved outline of the dorsal dulness in pleuritic effusion to which Damoiseau first called attention. It should be also borne in mind in any cursory examination for consolidation or for any other abnormality. You will now thoroughly understand that the immediate neighbourhood of the spine does not afford a fair opportunity for pulmonary percussion. The true pulmonary note should be sought some distance away from the middle line.

Putting aside slight variations in degree, the same spinal resonance is continued below the twelfth dorsal vertebra over the sacrum, and usually as far as the coccyx. But at the level of the true pelvis it is apt to be modified by the contents of the latter, or in pathological states to be changed into dulness.

The uses of spinal percussion at various levels.—In the neck the aid of percussion is not often required, neither is it often needed above the level of the third dorsal. The region between this and the twelfth yields the chief clinical results.

(1) From the third to the fifth or sixth, mediastinal, glandular, or other swellings, particularly œsophageal new growths and the secondary consolidation of the edge of the lungs due to them, may produce definite dulness of the spinous processes, and they always give rise beyond the lateral vertebral strip of resonance to a definite impairment of the pulmonary resonance.

(2) The heart's region included between the fifth and the tenth does not yield, putting aside exceptional cases, any dulness of the spines themselves.

(3) The hepatic region from the tenth to the twelfth likewise fails to present dulness of the spines, except in children.* But where there is effusion of fluid into the pericardium these spines become dull, as well as the surfaces adjoining them, chiefly on the left side. I have given to this dulness, which is a valuable confirmatory sign of, and indeed, in adults, a test for the presence of effusion, the name of "the lower dorsal dull patch."† You will have no difficulty in identifying its presence in all cases of even moderate effusion.

(4) Below the twelfth the spines are generally resonant on percussion, owing to the vicinity of gas-containing bowel. But their resonance may be impaired in some cases in connection with considerable solid deposits in contact with the vertebræ. I need not allude to percussion of the loin, which is commonly practised. The outline of the kidney, whether in health or disease, can with sufficient practice be easily made out; and this exercise should not be neglected by clinical pupils.

Percussion of the sacrum, and, in association with it, percussion of the iliac bones, has not been practised within my observation, and yet it may supply information not otherwise easily got. Intra-pelvic disease does not necessarily give rise to absolute dulness if there should be much infiltration of the sigmoid or of the rectum, yet it will often facilitate the diagnosis of early ascites, as I have elsewhere described. But here, again, the results are not absolutely conclusive.

The Pleximetric Resonance of Solid Viscera.‡

Organs usually recognised by their dulness may yield, wholly or in part, a resonant, nay a tympanitic note by playing the part of pleximeters to some contiguous resonant hollow viscus. Those which are entirely contained within the abdomen may still be recognised by palpation, but we are more liable to be misled in connection with those which

* Cf. "Remarks on the Dorsal Test for Pericardial Effusion: an Addendum," 'Brit. Med. Journ.,' Jan. 23rd, 1898.

† Cf. "Practical Aids in the Diagnosis of Pericardial Effusion, in Connection with the Question as to Surgical Treatment," 'Brit. Med. Journ.,' March 21st, 1896.

‡ Cf. 'Lancet,' July 2nd, 1898, vol. ii. p. 23.

are wholly intra-thoracic. The most frequent instances of this pleximetric condition occur in the organs adjacent to the stomach :

- (1) The spleen,
- (2) The left lobe of the liver, and
- (3) The heart.

A "boxy" spleen is not uncommon. As the note is almost always due to an inflated stomach (less often to inflation of the colon), very careful percussion of the costochondral basic area is requisite. The anatomical particulars of this region I need not dwell upon again. The "boxy" character of the splenic area will be easily distinguished from the resonance which arises immediately from the stomach itself. My first experience of this peculiarity was in a case of typhoid fever. The derivation of the abnormal resonance having been realised, a determination of the size of the spleen by its "boxy" note became quite as easy as it might have been by means of the usual dulness; nay, easier. Since then the experience has often been repeated.

The left lobe of the liver is so frequently pleximetric that this might almost be regarded as one of its normal conditions. It was long ago pointed out* that this provides us with an easy means of defining the boundary between the cardiac and the hepatic areas—a determination which it is always possible to make even when the left lobe is dull owing to the stomach being charged or quite empty. Almost invariably this section of the liver dulness is at least modified in the direction of resonance owing to the vicinity of the stomach, and therefore in percussing the absolute hepatic dulness from the extreme right towards the left a change takes place from absolute to partial dulness at the epigastric notch. Often the resonance of the left lobe becomes marked, but the "boxy" tympanitic note mentioned in connection with the spleen is not produced; and, moreover, the influence does not spread to the entire organ. A universal "boxy" note is probably produced either when a solid organ, such as the spleen, is in complete contact with a resonant surface larger than itself; or when a large organ, such as the liver, forms part of the wall of a strictly limited gaseous collection, con-

tained, as it were, in a box, and possessing some degree of tension. To this condition I have referred elsewhere.* That belonging to the left lobe of the liver is usually different, the tension of the gases in the stomach not being sufficient to change its note to a tympanitic one.

(a) Dr. Lennox Wainwright of Folkestone, informs me that he has noticed that in pleuritic effusion the resonance of the scapula is apt to interfere with a determination of the dulness due to fluid.

* Cf. 'Lancet,' *loc. cit.*

The Treatment of Gastric Ulcer by Large Doses of Bismuth.

Gastric ulcer has been treated by very many methods, some of which radically differ from one another, and the use of bismuth in this affection is by no means recent. Within the last few years, however, it has been suggested by Fleiner that good results follow the use of massive doses of this substance. This clinician employed as much as 300 to 450 grains of bismuth in suspension in water, poured into the stomach by means of a stomach-tube after previous lavage, and in his paper he referred to the experiments of Mattheys upon the action of bismuth in hastening the cure of experimentally produced ulcers in the stomachs of dogs.

At a recent meeting of the Manchester Therapeutical Society, Dreschfeld read a paper upon this subject in which he protested against the use of the stomach-tube as a method of administering these large doses of bismuth, on the ground that it is not wise to pass a tube into an ulcerated stomach. He stated that he had employed from thirty to fifty grains of bismuth subnitrate three times a day, suspended in water, and swallowed without the aid of a tube. Under these circumstances he found the condition rapidly relieved, vomiting ceased, and digestion improved, although he allowed light nitrogenous food, such as fish or fowl, to be given. Ultimately the ulcer healed. These large doses of bismuth have never, in his experience, produced constipation, but rather a slight tendency to pain and diarrhoea. Dreschfeld has also found this method useful in both acute and chronic cases, and also in acid dyspepsia with neurasthenic condition.

Therapeutic Gazette, June, 1898.

* "On the Value of Accuracy in Cardiac Percussion and Auscultation," 'Lancet,' August 29th, 1891. Also "Cardiac Outlines for Clinical Clerks and Practitioners, &c." With sixty-two illustrations. (London: Baillière, Tindall and Cox, 1892.)

LATERAL CURVATURE OF THE SPINE, OR SCOLIOSIS.

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THE study of scoliosis is, perhaps, best begun by the examination of a skeleton in which the condition is both typical and pronounced. By the

are—1st, an S-shaped lateral bending of the spine, to the right in the dorsal, to the left in the lumbar region; 2nd, a rotation of the vertebræ involved in these curves, their bodies being turned towards the convexity of the curves.

Seen from behind (Fig. 2) the same features are observed, and in addition it is seen that the spinous processes are rotated towards the concavity of the curves,—that is, in the opposite direction to the bodies. In slight degrees of the affection this rotation may be sufficient to bring the spines into vertical line with one another. This is a matter of

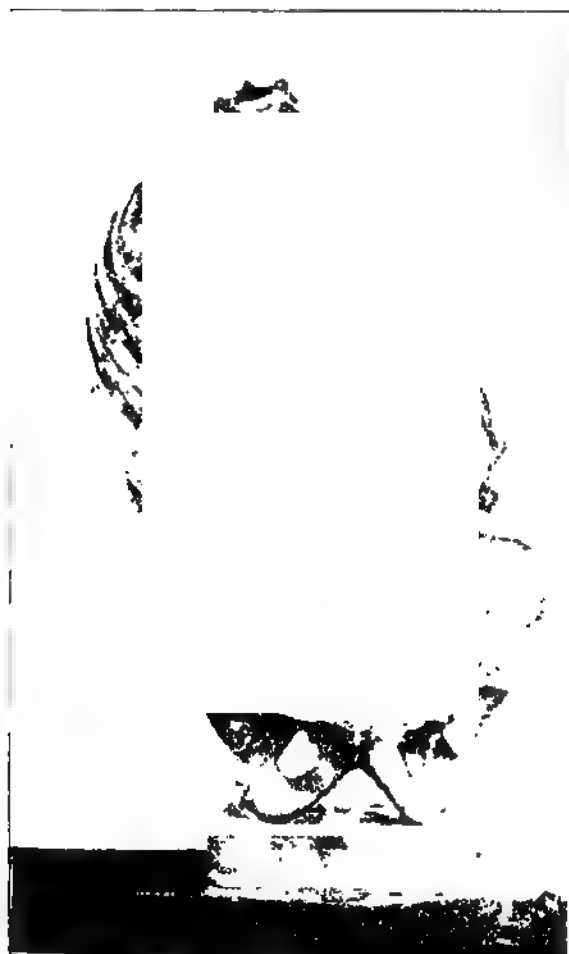


Fig. 1.—Front view of a scoliotic skeleton in the Museum of the Royal College of Surgeons.

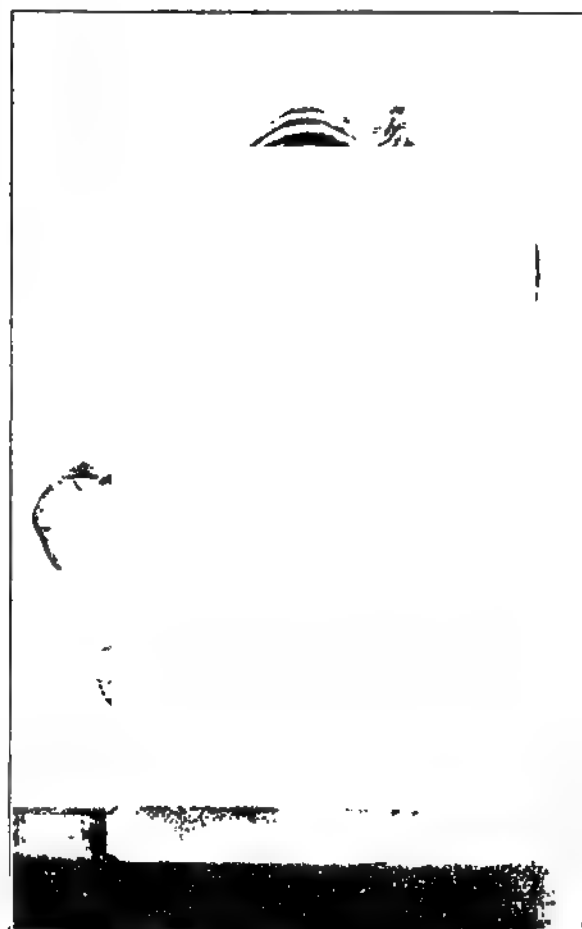


Fig. 2.—Back view of the same skeleton.

kind permission of the President and Council of the Royal College of Surgeons, I am able to reproduce three views of such a skeleton. Seen from before (Fig. 1) the chief features observed

practical importance in diagnosis, for there may be present a considerable amount of scoliosis without any deflection of the spines. In severe cases the rotation of the spines in the opposite direction to

the bodies is not so marked as might be expected. This is due to the resistance offered to the progress of the distortion by the attachment of muscles to the spinous and other processes, which induces a structural asymmetry in the individual vertebræ. Another striking feature, seen both in the anterior and posterior views, is a general inclination of the trunk to the right. This deviation of the trunk is often seen even in slight cases of scoliosis; an instance is given below. It will be

dorsal aspect of this typical scoliotic skeleton, it may serve to lend reality to the study if a rough attempt at restoration of the person to whom the skeleton belonged is made, as in Fig. 3.

Turning next to the side view of the skeleton, the alteration in form is not less remarkable. The most pronounced change is a sharp forward bend in the dorsal region; this *kyphosis* usually precedes the lateral deviation. It gives rise to a stoop, and young persons in whom this peculiarity of carriage



Fig. 3.—Restoration of the back, from the skeleton shown in Figs. 1, 2, and 4.

further observed in the dorsal region that the ribs on the convex side are separated from one another, and are sharply bent at their angles, their posterior parts having followed the rotation of the vertebræ, whilst their anterior parts have resisted this movement. On the concave side the ribs are close together, and are displaced forwards by the rotation of the vertebræ.

Before passing beyond the consideration of the



Fig. 4.—Side view of the same skeleton.

is noticed should be made to abandon it as speedily as possible, and if they fail to do so they should be examined for scoliosis.

The examination of the patient.—The natural flexibility of the spine and the varying inclination of the pelvis in standing render it advisable that the patient should sit during the examination. In this way the amount of actual deformity in the spine, as distinguished from temporary deflections arising

from inclination of the pelvis, due to standing "at ease," or to inequality in the length of the legs, can best be ascertained. If the deformity entirely disappears when the patient sits down, it may be concluded that there is no scoliosis, but merely a temporary deviation due to inclination of the pelvis.

Fisher and some other authors claim that distinction should be made between lateral "deviation" and lateral "curvature" of the spine. On this point Bradford and Lovett observe, "this distinction is not always a practical one, as in the early stage of lateral curvature, before fixation has occurred, permanent rotation is not always recognisable."

When the patient is seated squarely on a firm stool, and the whole of the back is bare, a rough

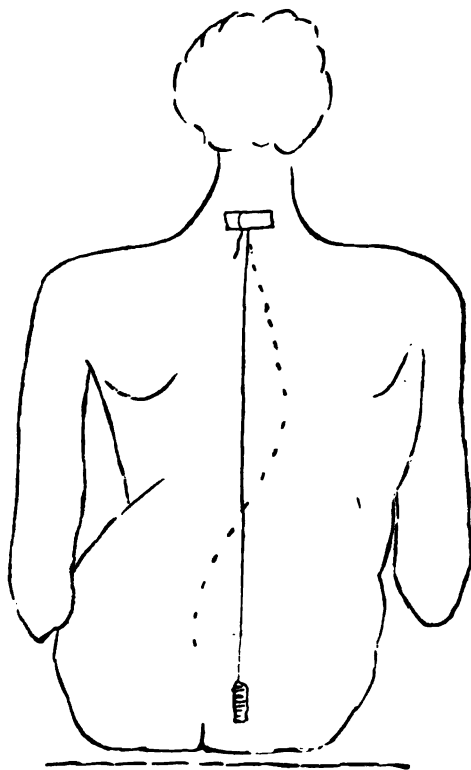


Fig. 5.—Sketch of the back of a young woman, aged 15, who was first noticed to walk "awry" two years previously.

sketch of the back (Fig. 5) should be made, and various features that cannot be shown in the sketch, *e. g.* the hollowness of the loins, should be noted. The degree of inclination of the trunk may be

measured by fixing a plumb-line to the vertebra prominens by strapping, and noting the distance between the plummet and the top of the intergluteal cleft (see Fig. 5).

The amount of dorsal rotation is best seen when the patient stoops with the arms folded across the chest, and may be recorded by moulding a strip of lead or tin transversely to the back at the level of the angles of the scapulæ, the position of the latter and of the spine being marked on the strip before it is removed. The curve thus obtained can be transferred to paper by tracing with a pencil along the inner surface of the upper edge of the metal. A tracing of the kind is shown in Fig. 6. A similar

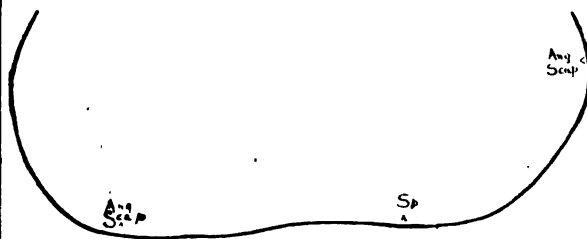


Fig. 6.—The contour of the back obtained from the patient shown in Figs. 5, 7, and 8.

tracing, taken between the twelfth ribs and the iliac crests, may be used to record the lumbar rotation (Roth). Photographs alone do not give a complete record, but they serve to check the graphic notes made in the sketch. Two photographs should be taken, one a full back view (Fig. 7), and one of the body in profile (Fig. 8). The front of the chest should be inspected. In a case of right dorsal curve the right side of the chest is flattened, whilst the left side is prominent, as shown in Fig. 9.

With a view to prognosis the degree of resistance to manual correction offered by the spine should be noted.

Finally, the patient should be examined standing, in order to ascertain whether or not there is a *mal-position* added to the permanent *deformity* already observed.

The more elaborate methods of recording degrees of lateral deviation and rotation, *e. g.* Barwell's scoliosimeter, are, in spite of their great ingenuity, difficult of exact application, because the bony prominences are not *points* but *surfaces*, which cannot be marked twice on exactly the same spot; moreover the skin covering them is free to move,

and hence a mark made over a spinous process may, by a slight movement of the body, be found on a second trial to be at some distance from it.

It is of importance to ascertain the patient's height at the beginning, and from time to time during the course of treatment. The height is diminished by the twisted form of the spine, and under successful treatment it increases more rapidly than the natural growth of the body would account for.

in too rapidly growing children, and in those suffering from rickets and rheumatoid arthritis, obliquity of the pelvis in standing, and oblique and twisted postures in sitting, readily determine scoliosis. In rickets, again, nasal obstruction from adenoids, or respiratory impediment from bronchitis, not only produce pigeon-breast and other deformities of the front and sides of the chest, but also cause distortion of the spine, which is the main stem of the thorax.

Fig. 7.—A photograph of the back of the same patient.

The causes of scoliosis.—Any posture assumed too frequently and persisted in too long is liable to cause deformity even in healthy parts. Young and healthy individuals are hardly likely to remain voluntarily in one position for a sufficient length of time for deformity to arise; but if the resisting power of the bones or ligaments or both is diminished, or if the strength of the muscles is impaired, then, in a long, unstable, jointed column, as the spine is, deformity is easily produced. Thus

Fig. 8.—Photograph of the side view of the same patient.

Infantile paralysis, so common a cause of deformity in the extremities, is also responsible for lateral curvature more frequently than is generally suspected.

The pathology of scoliosis is, perhaps, best appreciated by comparing it with deformities of similar origin but of more simple character, such as genu valgum, which like scoliosis occurs chiefly in rickets and adolescence, and which is also met with in infantile paralysis. A most careful examina-

tion of a case of genu valgum, that of a girl aged seventeen, the deformity beginning two years before death, and therefore a case of genu valgum adolescentium, has been published by Prof. Johnson Symington in the 'Transactions' of the Royal Academy of Medicine in Ireland, 1896, and by the courtesy of the author and of the Council of the Academy I am allowed to reproduce in Fig. 10 the appearance of the bones in this case. On reference to the figure it will be seen that the epiphyseal cartilage at the lower end of the femur, and in a slighter degree that at the upper end of the tibia, show the changes characteristic of rickets, changes that render easy of comprehension the deformity seen in the bones at the knee-joint, of which the clinical expression is genu valgum. With this

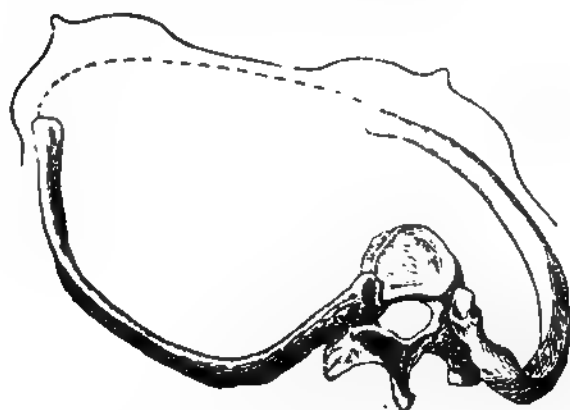


Fig. 9.—A vertebra and ribs, showing the transverse deformity of a segment of the thorax, with the outline of the breasts suggested.

analogy before us the question naturally arises whether there is present in scoliosis of adolescence some change in the bones, similar in character to that seen in this case of genu valgum. My own opinion is that such is generally the case, and this opinion has previously been expressed by Volkmann in the phrase *rachitis adolescentium*. Experience has led me to believe that the cases of scoliosis commencing in early childhood and directly attributable to rickets are far more numerous than most authors appear to think. Although in the more severe grades of rickets extreme distortion may ensue, and so lead the observer to conclude that rachitic scoliosis is something essentially different from the scoliosis of adolescence, yet if a large number of cases are carefully observed, it will be found that in the slighter grades of rickets

the resulting scoliosis conforms to the regular type described above; and, further, that the more closely the evidence of the date of the deformity is inquired into, the earlier will this be found to be, so that the cases of scoliosis first observed in

Fig. 10.—Section of the bones at the knee-joint in genu valgum (Symington).

infancy and those first noticed at puberty are connected together by other cases arising at any age between one and fifteen years.

Even among the children of the upper classes a case of adolescent scoliosis, if carefully examined,

will frequently show traces of rickets in infancy by the presence of slight tibial and other deformities. This, together with the general condition of the patient, has often led me to the conclusion that in many such cases there is a recrudescence of rickets during adolescence. In other words, such cases of scoliosis are due as much to the softening of bones and ligaments as to muscular weakness and inequalities in the lower limbs. Seeing, however, that paralysis alone is capable of producing scoliosis, the importance of muscular weakness in its causation must not be overlooked.

The hereditary character of scoliosis.—All who have to deal with many cases of scoliosis will have observed families in which several members are affected, and in which the deformity occurs in different generations. Excepting rare cases in which the deformity depends on some inherited peculiarity, such as a half-vertebra, it is not difficult to arrive at a conclusion as to the nature of this hereditary trait. It is experienced in families, the members of which are tall and slim, during the period of rapid growth, and sometimes there is in the family a tendency to rickets or to rheumatoid arthritis, affections which diminish the resisting power of the skeleton. Thus the hereditary character of the deformity is nothing more than a common lack of stability in the spinal column during adolescence. It will be noticed that where several sisters are affected, the direction of the deviation is often reversed in the different individuals.

Diagnosis.—In severe cases it might be thought that there would be little difficulty in recognising the condition at a glance, yet the prominent vertebræ in high dorsal curves have been mistaken for tumours in the supra-clavicular fossa; prominent lumbar curves have also been mistaken for growths. Adams has recorded a case in which the lumbar prominence was mistaken for an abscess. I have known surgeons of experience, misled by the prominence of one hip, to ascribe the deformity to some abnormality of the hip-joint. A little clinical experience and study of scoliosis will, however, be sufficient to prevent mistakes in pronounced cases. It is in the earlier stages of the deformity that diagnosis is of the greatest importance. The average appearance of a case of moderate degree, that most usually seen at a first visit, is shown in Fig. 11.

The features already described in more severe cases will be readily recognised; among them the oblique carriage of the body may be emphasised. In all the cases so far referred to the dorsal curve has been convex to the right, the lumbar to the left. Cases in which the curves are reversed are by no means uncommon, and the foregoing description will apply equally to them by merely transposing the words "right" and "left." By

Fig. 11.—The back of a boy aged 7 years, showing early scoliosis. The trunk is inclined to the right, the left hip projects, and the lateral deviation is seen in the dorsal region.

the time that the mother, the nurse, or the dress-maker has noticed an "outgrowing" shoulder or hip, or some peculiarity of carriage, the deformity is generally well advanced. Cases which in my experience present most difficulty are those in which what may be termed *intermittent scoliosis* is present. At one visit the patient may be found to present lateral deviation, rotation, and inclination of the trunk; the next day, perhaps, the same patient may be carefully examined, and no trace

of scoliosis be found. This condition is observed chiefly in boys with long weak backs.

Tuberculosis of the spine, both in the early and late stages, may cause a localised lateral deviation of the spine; but the rigidity, pain, &c., that characterise tubercular disease, as well as the localised nature of the deviation, will serve to distinguish this condition from scoliosis.

Rigidity, however, is the most important sign of tubercular disease.

If all the points in the method of examination described above are carefully studied, angular curvature, even in a slight degree, will readily be recognised. It must always be borne in mind that in the cervical and lumbar regions diminution of the normal concavity backward is the equivalent of a certain degree of angular deformity in the dorsal region.

Prognosis.—Scoliosis, when once established, is never *spontaneously* cured, or even diminished. The progress of the deformity is, however, not infrequently *arrested* by improvement in the general health of the patient. If such arrest occurs before much distortion has been produced, only a slight deformity, recognised chiefly by the tailor or dress-maker, will remain, and will possibly give no farther trouble. But in a certain proportion of cases, after remaining stationary for many years, the deformity again begins to increase, owing to the development of rheumatoid arthritis, or some other disturbance of nutrition.

Unfortunately we cannot rely upon an early arrest of the deformity in any given case, for in many instances the disease increases steadily until a shocking deformity is produced. Nor are the disabilities experienced by those who suffer from severe scoliosis due merely to altered personal appearance. The alteration caused in the position of the heart and lungs produces considerable dyspnoea. Neuralgic pain in the spine is not uncommonly complained of, especially where rheumatoid arthritis is present.

A slight lumbar curve in a young child, if left untreated, may increase in severity so that the patient's capacity for walking and movement is greatly diminished.

B. Roth states that in every case of scoliosis a certain posture can be found in which the deformity is reduced to a minimum. This posture, he says, gives the "key-note" in prognosis, being an

indication of the amount of correction possible. A recent writer, P. G. Lewis, says on this point, "Taking the most common key-note position, viz. that with the left arm up by the side of the head, and the right out at right angles with the body, one cannot but wonder where the key-note is. The appearance of deformity is thus removed, chiefly by putting the lower part of the trapezius and the upper part of the latissimus on the stretch on the left side." My experience is in harmony with this statement, since I have not found the so-called "key-note" of any practical value.

A few practical points considered as elements in prognosis may be mentioned. (1) The general type and stamina of the patient and of the patient's family. (2) The degree of deformity present. (3) The duration of the deformity. (4) The character of the curves. (5) The patient's age. Further, a lumbar curve is more difficult to correct than a dorsal one.

Information as to the probable degree of correction possible in any given case is obtained by trying to what extent the deformity can be diminished by manual pressure. It has been recently stated* that "osseous deformity of the vertebræ, even to the slightest extent, is to that extent incurable." In judging of the value to be attached to this statement it is to be remembered that up to the age of twenty-five years the vertebræ contain a considerable amount of cartilage, and that up to this age the body of a vertebra presents a structure comparable to that of a long bone. In describing the causation of scoliosis it was observed that any posture that entailed a lateral bending of the spine would, if maintained too long, produce scoliosis. This may be postulated from the known fact that the growth of a long bone is readily influenced when one side of the epiphysial cartilage is made to bear more than its share of pressure, *e.g.* in the production and in the cure of genu valgum. It follows that if in a young subject we can reverse the forces that act on the spine in the production of a scoliotic curve we can cure bone deformity in the spine, as we can in the bones at the knee-joint. By the use of effective apparatus employed continuously the growth of the vertebræ can also be guided. If this were not so the most funda-

* B. Roth, 'Brit. Med. Journ.,' October 9th, 1897.

mental principles of orthopædic surgery would be violated. The statement is, then, not correct in so far as it applies to growing subjects. After the growth of the patient has ceased, and in relation to certain methods of treatment, the statement holds good.

Treatment.—The question, "Does every case of scoliosis require treatment?" may be asked. For answer it is sufficient to recall the fact that there are not a few active and even athletic individuals between twenty and thirty who suffer from scoliosis, and in whom the deformity neither increases nor interferes with the activity of the person, the only symptom of the disease consisting in the deformity and a quickened respiration. After the growth of the bones has attained its full development, and the epiphysial cartilages have disappeared, a general supervision at long intervals is all that is required. Excessive exercise, which in the presence of impeded respiration is likely to produce cardiac dilatation or hypertrophy, is to be avoided. The onset of rheumatoid arthritis is to be watched for.

Preventive treatment.—In the physical education of children the possibility of the occurrence of lateral curvature is to be thought of. By suitable diet and general hygiene, rickets and bone weakness in general is to be guarded against. Attention to dress is important. It has been rightly pointed out that the dress and under-garments should be cut so that they are not tight across the chest when the wearer stands upright with the back to a wall. During the school period ample and proper food and exhilarating general exercises are to be secured. No one who has noted the effects on recruits of the physical drill as described in the official "infantry drill" can doubt the advantage of systematic drill upon the young. There is, however, a caution to be observed. Latterly in London it has become fashionable to submit young girls to fatiguing calisthenic and dancing lessons, lasting for two hours with but little intermission. Such classes are responsible for many cases of deformity. On an average half an hour of such exercise is sufficient at one time.

Threatened scoliosis.—When a tendency to scoliosis is detected special precautions are needed, *e. g.* proper school chairs and desks* are to be used;

* The "Clendenning" chair and desk answer all purposes for school use.

and the one-sided exercises, and those which like bicycling tend to promote stooping, are to be discarded. Increased intervals of rest on a proper chair or couch are to be observed, and the physician is responsible for the correction of inequalities of the legs and bad postures, also for the correction of errors of vision by suitable glasses.

The treatment of rickety scoliosis.—I am so constantly told by the mothers that scoliotic infants were crooked in the back when they were born, that I cannot avoid the conclusion that a certain proportion of cases of rickety scoliosis are of intra-uterine origin, and that in such cases treatment should commence with life. The infant should rest in Adams's padded wicker cradle, and the padding may be so arranged that it tends to correct the deformity.



Fig. 12.—Sketch of a mother carrying a child on the left arm, showing how the spine has in this posture a right dorsal and a left lumbar convexity.

The method of carrying an infant on the left arm usual with mothers and nurses should be avoided. The sketch, Fig. 12, was taken in the out-patient room. The child, aged 2 years, had rickets and marked scoliosis.

As soon as the child begins to sit up, a moulded

leather backboard should be substituted for the cradle. In rickety children of two years and over I find that the best results are obtained by the use of a Chance's splint. The splint must be removed twice a day, and the back should be well rubbed and moulded by the hands. The splint should be readjusted from time to time as the shape of the back improves. It is especially in rickety children that adenoids should be looked for, and, if found, completely removed. In paralytic scoliosis the only means of diminishing deformity lies in the application of a suitable support.

The treatment of actual scoliosis in adolescence.—

The measures at the command of the surgeon fall into three categories: 1, correctional postures and exercises; 2, instruments; 3, forcible correction. Before describing these at length it may be premised that the dietetic, hygienic, and medicinal treatment of the patient must be carefully managed in every case, and, further, whatever the special treatment the importance of general gymnastics as a hygienic factor has been recognised in all civilised countries. Special medical gymnastics as remedial agents have also been long established in orthopædic surgery. The measures most in use at the present time for lateral curvature can be traced to the system evolved by P. H. Ling in Stockholm between 1805 and 1839. The chief peculiar feature of this system is thus described by Busch:—"In the compound movements an alternate influence is exerted by both patient and gymnast, for either the patient performs a movement to which the gymnast opposes an equal resistance, in such a manner that he does not completely hinder this movement, but only renders necessary a greater degree of power to effect it; . . . or the gymnast conducts the movement of the patient's limbs, while the patient opposes the movement in accordance with his strength."

Ling's system, which was introduced into England by the late M. Roth, became needlessly complicated, but the principle of resisted movements remains as a valuable resource in orthopædic surgery. More recently G. Zander devised a system by which the gymnast in Ling's system is replaced by mechanical contrivances. B. Roth has applied Ling's system to the treatment of scoliosis. Of the first series of twelve exercises eight are performed with the patient lying on the back upon a firm couch, whilst the surgeon exercises in turn the

upper and lower limbs; three of the twelve exercises are directed to increasing the power of the spinal muscles. During most of the exercises the patient is directed to count aloud, with a view of exercising the respiratory muscles. An interval of two or three minutes is allowed between the various exercises, and during this interval the patient rests upon a special couch, the back resting on an upholstered board fixed at an angle of 75°. Later more severe exercises are used. It will be observed that this system is really a combination of mechanical with gymnastic treatment; the couch acts as a splint to the back. In M. Roth's hands this mode of treatment has had a measure of success. The drawbacks are, however, considerable. The author candidly admits that it is incapable of correcting bony deformity. In most cases of simple deformity, such as knock-knee, the bones are affected from the beginning; and when scoliosis is not corrected spontaneously when the patient lies down, it may be inferred that alteration in the form of the bones is also a factor in producing this deformity. And in such cases the so-called "treatment by exercises alone" may result in stopping the progress of the malady, but not in curing it. The amount of time taken up by daily exercises requiring the personal supervision of the surgeon renders the method very costly, and interrupts schooling.

In bad cases the special reclining couch and rest in the horizontal position are, in my experience, far less effective than a properly constructed and properly managed Chance's splint combined with regular exercises. It is especially in fairly muscular youths that the power of will and muscles to arrest deformity is most effective, and the use of even simple instruments is to be avoided if possible. By the use of elastic bands, after a few drills under the supervision of the surgeon, the need of daily visits is avoided, though it is of course necessary for the surgeon to inspect the patient at regular intervals in order to see that the case is doing well. I need not detail the exercises for the muscles of the limbs, but I may briefly refer to the chief of those I employ to exercise the muscles of the back and abdomen, and those for increasing respiratory power. Flat-foot, if there is any tendency to it, should also be treated.

1. *Extensor muscles of neck.*—Patient seated

in an ordinary low-backed chair, and an india-rubber tube fixed at its free end to the front of a webbing band which encircles the head. Hyperextension of cervical and upper dorsal spine repeated ten times.

2. *Abdominal and ilio-psoas muscles.*—Patient lying on the back with the arms extended above the head. The body is raised to the sitting posture. Repeat five times.

3. *Erector spinæ.*—Patient lying on the face, arms out at right angles, hands prone, face turned to the side of dorsal convexity. Patient supinates hands, throws the scapula well back, raises the hands from the floor, and lifts the trunk. Repeat three times. (R. H. Sayre.)

4. *Dorsal rotation and respiration.*—Patient seated as in 1. The arm on the side of the dorsal convexity is passed in front of the body, the other behind the body, the hands holding elastic traction cords. Slow, deep respiration. (Barwell.)

5. *Lumbar rotation, &c.*—Patient standing; head, shoulders, and heels touching the wall. The pelvis is rotated away from, whilst the trunk is inclined towards the lumbar convexity.

The patient should be taught to breathe deeply and regularly during all exercises, a habit that can be acquired by counting aloud.

Special resistance exercises for the chest.—The mother or nurse can be taught to exercise general resistance to inspiration with the outspread hands, whilst the patient sits with the back supported in a suitable chair, and breathes deeply and regularly.

Instrumental treatment.—Bearing in mind that a certain proportion of cases of spinal curvature are due to rickets and a still larger proportion to conditions which are closely akin to rickets, there is a clear indication for the use of instruments wherever bony deformity is present. In the choice of an instrument the objects aimed at must be kept closely in mind. The instrument should be firmly fixed to the pelvis; the main upright should have the outline of the normal slight dorsal and lumbar curves; the splint should be so applied that the dorsal excurvation (kyphosis) and the lumbar incurvation (lordosis) are corrected. Pads should be placed so that they tend to correct both the lateral deviation and also the rotation. Whilst giving support to the spine, and thus resting the spinal muscles, it should leave the

latter free to act within a limited range. The instrument should be arranged in such a manner that its form can be easily modified to follow up improvement in the shape of the body. It should be as light as is compatible with efficacy, and it should be easily removable by the patient or mother for the performance of exercises. Another desideratum is that it should be moderate in price. It should not interfere with the patient's wearing ordinary dress and mixing in society, going to school, and taking ordinary exercise. Such an instrument acts like a simple splint in such a condition as genu valgum, but the complexity of the deformity in scoliosis demands a corresponding complexity in structure.

The instrument that I have found of the greatest service in scoliosis is Chance's splint. The details of this instrument and its structure and mode of action I have described in a former number of this Journal (March 9th, 1898). If wrongly used the instrument, simple as it is, may do harm instead of good. At the commencement of treatment it is most important that the splint should be removed twice a day, and the back massaged. In the case of children the spinal exercises mentioned should also be done twice daily, whilst general exercises for legs and arms should be performed with the instrument applied. The instrument takes the place of the special couch used in following the method of treatment without instruments, and it is much more effective. In most cases the instrument should be worn at night. The marked and rapid improvement in the shape of the spine I have observed in young subjects treated with Chance's splint has caused me to relinquish a credence I formerly lent to the words of those who denounce the use of any instrument in scoliosis. It is the abuse of instruments that is to be deprecated, not their use. The conditions are similar to those present in a case of genu valgum. The application of the force is more difficult, but rightly applied in a suitable case a similar effect is produced. Arbuthnot Lane* has observed, "in rickets . . . the epiphysial line, which is large and irregular in outline, reacts to pressure in a much more marked manner than does the epiphysial line of the child which is merely wanting in vigour." A close watch for the

* 'Clinical Lectures on Surgical Subjects,' 1898.

onset of scoliosis should be kept in rickety children—and rickets is by no means confined to infancy, and as soon as the condition is observed continuous mechanical support is required. The number of different appliances is so very great that I have not tried them all; I have, however, seen many different kinds, and, as above stated, Chance's splint, which itself is an improvement on one made in a former generation by Sheldrake, is the only one that I can rely on as having a curative power. Instruments that depend for their effect on axillary crutches, or in any way impede respiration, also rigid and cumbrous corsets of poroplastic or plaster of Paris, are to be avoided.

Barwell, deprecating without discrimination the use of any rigid instruments, recommends amongst other measures the use of elastic swathes. For a typical case, one bandage acts from the lumbar convexity to the top of the opposite trochanter, and would doubtless either abduct the corresponding thigh or tend to diminish the lumbar convexity. A second surrounds the right shoulder and joins the upper part of the loin bandage, both in front and behind. This swathe is supposed to counteract the dorsal curve, but from its disposition the force exerted by it must have a downward as well as a horizontal action, and so tend to increase deformity at the junction of the curves, and it also would tend to increase the dorsal rotation. This last feature has evidently struck the author, who arranges the bands in a different manner when dorsal rotation is a marked feature. The imperfect mechanical effects of the dorsal part of this apparatus render it in my opinion an uncertain means of treatment.

Forcible correction.—There is no doubt that in severe cases in growing subjects forcible correction combined with exercises, and the continuous application of an effective splint may, in suitable cases, give a rapid improvement in the form of the spine. Lorenz effects this end by bending the patient with the convexity of the dorsal curve resting on a padded horizontal bar; Barwell by strong and wide webbing bands acted on by pulleys. Noble Smith has lately recommended manual correction. I employ both the latter methods in suitable cases.

The patient.—One last observation may be allowed; that is, whatever the deformity may be, in selecting the exact method of procedure the strength, age, and sex of the patient must be con-

sidered. Treatment suitable to a muscular boy would not be appropriate to a delicate girl; and any energetic gymnastic treatment would be out of place in the case of a patient suffering from advanced heart disease or phthisis. There is no affection that requires more thought, more careful selection of methods, than lateral curvature of the spine. Moreover, in the course of the same case the treatment requires variation as the condition improves.

The Compression Treatment of Pulmonary Tuberculosis.

—Dr. Murphy proceeds upon the assumption that a tuberculous lesion of the lung, like one of a joint, for example, may ordinarily be healed quite readily by securing immobility—functional rest of the affected part. In pursuance of this idea he immobilises the lung by compressing it, crowding it back upon its hilum, establishing a sort of artificial atelectasis. This he accomplishes by injecting a quantity of nitrogen into the pleural sac. Nitrogen, he finds, neither exerts any untoward effect upon the pleura, nor is absorbed to any appreciable extent; it simply keeps the pleura distended and the pulmonary tissue compressed. It is said that during the continuance of this compression the patient feels remarkably free from the symptoms that had previously preyed upon him. The gas is allowed to remain in the pleura for a period of several weeks, and then it is withdrawn. In a goodly number of instances the symptoms do not return, and the inference is drawn that the disease has been overcome. The lung again becomes aerated, and expands almost, if not quite to its normal size. If on the removal of the nitrogen the morbid symptoms return, more of the gas is thrown into the pleura and kept imprisoned there for another term of weeks. It is said that this second injection is by no means always found necessary, and that when it is called for it almost invariably suffices for the cure of the disease in that lung. Then the other lung is treated in the same way.—*New York Medical Journal*, June 25th, 1898.

The Transition of Fat into the Milk of Nursing Women.

—Bendix finds that food rich in fat does not increase the percentages of fat in the milk of nursing women, and that the latter is probably derived from albumen.

ON NERVOUS VOMITING.

BY

G. BERTRAM HUNT, M.D., M.R.C.P.

CASES of nervous vomiting, that is to say vomiting for which no organic cause can be detected during life, are sufficiently common; but it is certainly rare for such a condition to lead to a fatal issue, thus allowing the diagnosis to be verified by a post-mortem examination; and for this reason the following case is of interest. A married woman, aged twenty-nine, was admitted on January 25th, 1897, into University College Hospital, under the care of Dr. Roberts, the history being as follows:—Twelve months previously she had an attack of abdominal pain and vomiting which lasted for several days, and then left her entirely. At the end of October, 1896, she had a severe "bilious attack," and on the following day vomited a tea-cupful of bright blood. From this day to the time of admission she constantly vomited directly after meals, and also between meals; there being occasionally a small quantity of blood in the vomit. When admitted the patient was but slightly emaciated; the abdomen was rigid and tender all over, but she referred the spontaneous pain entirely to one point in the epigastrium, pointing to the painful spot with one finger in the way described in the text-books as typical of gastric ulcer. The treatment consisted of cutting off all food by the mouth, and feeding entirely by nutrient enemata; fomentations and poultices to abdomen; morphia hypodermically, while tincture of iodine, ipecacuanha wine, and tincture of opium in small and frequent doses were all tried successively by the mouth. In spite of treatment the vomiting continued almost incessantly for five days, being so continuous as to almost prevent sleep; the vomit contained bile, and occasionally small quantities of dark blood. The general nature of the case with the history of hæmatemesis of course strongly suggested gastric ulcer; but as the patient was extremely emotional, and the vomiting was unusually frequent, and did not respond to treatment applicable to gastric ulcer, it was suggested that there was a marked nervous element to be dealt with. The patient was accordingly put on drachm doses of potassium bromide by the rectum

four times a day, and in thirty-six hours the vomiting, which was previously increasing in severity, had entirely ceased; and in four more days she was successfully taking solid food by the mouth, and in a fortnight was discharged cured.

She remained in good health for a year, when the pain and vomiting quite suddenly returned, and she was readmitted on January 28th, 1898. The symptoms were exactly the same as in the previous year; the vomiting being almost incessant, the vomit consisting of clear bile-stained liquid, frequently containing traces of blood. In this attack, however, the treatment by rectal feeding, bromide, and morphia, was absolutely inoperative. The vomiting increased, the pulse became rapid and feeble, and in twelve days the patient died, the end being accelerated by an attack of uncontrollable diarrhoea with offensive stools.

The post-mortem revealed absolutely no lesion except a catarrhal inflammation of the lower end of the ileum, which, no doubt, accounted for the terminal diarrhoea. The stomach was perfectly healthy, except for a few submucous hæmorrhages. There was no ulceration, erosion, or atrophy of the mucous membrane. The liver, kidneys, and other organs were all healthy.

The diagnosis of such a case during life presents many difficulties. In considering cases of severe vomiting one must of course follow the general rule of excluding all organic diseases before venturing to diagnose a functional condition, and the organic disease producing long continued vomiting may be either in the stomach itself, or in some other organ. In the present case, as not infrequently happens, the symptoms very closely resembled those of gastric ulcer, although no trace of a recent or even a healed ulcer was to be found post-mortem. The difficulty in the diagnosis between the two diseases is increased by the fact that they are both met with most frequently in the same class of patients, viz. females under the age of forty. It is especially noticeable how much hæmatemesis may occur in those cases, presumably from rupture of capillary vessels from violent strain while vomiting.

A few years ago a young girl, who presented all the typical symptoms of gastric ulcer, died in Dr. Ringer's ward with profuse and repeated hæmatemesis, the stomach being found to be quite healthy after death. The fact that patients not infrequently

die with all the symptoms of ulcer of the stomach, no lesion being found on examination, naturally raises the question whether many more cases which are diagnosed as gastric ulcer, and then recover, are not really examples of simple nervous vomiting without organic lesion, so that in the diagnosis of gastric ulcer, besides the well-known fallacy of failing to recognise the condition owing to the latency or absence of symptoms, we may also fall into the opposite and less recognised error of diagnosing ulcer when it is absent. The points indicating a simple functional disturbance are sudden onset of the symptoms, inordinate frequency and severity of vomiting, which is frequently not associated with food and not accompanied by nausea, absence of emaciation, prominence of emotional or other nervous symptoms, such as globus, craving for food, hysterical polyuria, or neuralgic affections of other parts of the body, and sudden cessation of the vomiting with or without treatment. Pressure on the abdomen in the cases of hysterical vomiting usually relieves the pain, while it frequently increases the pain of ulcer. Leube lays much stress on the fact that we can frequently remove neurotic gastric pain by the application of the constant current to the abdomen, while the pain of ulcer is never relieved in this way.

Having eliminated gastric ulcer, which is the only organic disease of the stomach itself likely to produce vomiting of this nature, there remain, however, certain affections of other organs which must be excluded before the diagnosis of functional vomiting can be made. The chief diseases which may reflexly produce long-continued and severe vomiting, often very difficult to recognise during life, but satisfactorily eliminated by the autopsy in the present case, are phthisis, anæmia, Bright's disease, movable kidney, Addison's disease, cerebral tumour, tabes, and early pregnancy, in any of which conditions vomiting may be a prominent and, indeed, the only symptom. Another condition, first described by Bristowe ('Practitioner,' p. 101, 1883), which accounts for some cases of vomiting of a so-called hysterical nature, is chronic dilatation or spasm of the œsophagus. It is very important to recognise this condition as it may be readily treated and cured by feeding through an œsophageal tube; but in the present case, as in the majority of instances, the vomit was strongly acid

in reaction, and undoubtedly came from the stomach, not from the œsophagus.

Having concluded that the derangement is a functional one, it must next be considered to which of the many varieties of nervous derangements of the stomach the affection belongs. In the first place these cases of simple nervous vomiting must be distinguished from cases of anorexia nervosa described by Trousseau, Gull, and others, for in the latter condition the primary defect is entire loss of appetite, with consequent severe emaciation, vomiting being absent in most cases, and in none being a prominent symptom. In other cases the vomiting has been considered to be not merely due to a primary derangement of the neuro-muscular mechanism of the stomach, but to be secondary to some alteration in the secretion of the gastric juice. In many of the cases an excess of hypochloric acid is found, but this alone would be probably insufficient to produce severe vomiting. A peculiar condition, in which there is continuous secretion of the gastric juice in the intervals between meals, has been described by Reichmann, who, in 1887, had seen sixteen examples. In this affection, which may be either periodic or continuous, the gastric secretion collects in the stomach, and is vomited in the morning, and occasionally through the day. But usually in these cases of nervous vomiting, as in the present instance, the vomiting during the attack is almost incessant, and is not likely to be due merely to an alteration in the secretion of the gastric juice.

Two other theories which have been advanced to explain these cases are that of Bouchard, who considers the primary change to be atonic dilatation of the stomach, followed by putrefactive food changes, with consequent absorption of toxic products, producing the vomiting and other symptoms; and that of Glenand, who considers the perverted gastric contractions to be due to a prolapse downwards of the stomach, the so-called gastropptosis, which is frequently associated with dropping of the other viscera. These theories obviously fail to explain the present case as neither dilatation or dropping of the stomach was found at the autopsy; and although these conditions occasionally exist yet they probably do not explain the cases described clinically as nervous vomiting, as they have not been found in the few post-mortems on these cases. As all the theories which have

been considered, namely, continuous secretion of the gastric juice, dilatation of the œsophagus and stomach, and dropping downwards of the stomach, only account for a small minority of the cases of nervous vomiting, we must consider the condition to be due to a functional derangement of the nervous mechanism of the stomach, so that repeated spasmodic contractions, both of the walls of that organ, and of the other muscles concerned in the act of vomiting, are produced by slight causes which in health would be inoperative. Thus a slight indiscretion in diet, fugitive gastric catarrh, which in a healthy person would cause few or no symptoms, may in a hysterical patient with a perverted nervous system set up most excruciating pain or profuse vomiting. The prognosis of these cases of functional vomiting is usually good. The severity varies between rare attacks of periodic vomiting, and incessant vomiting lasting for a long time, as a case described by Dr. Martin, in which it occurred after every meal for periods during three years. But it is noticeable that even in these severe cases wasting is not pronounced, showing that there is no defect in the digestion or absorptive powers, and recovery is the rule. However, it must be remembered that death, as in this case, may occur, and a few similar instances have been recorded by different observers; so that although a fatal result doubtless suggests that the diagnosis during life has been mistaken, and that there may be some undetected lesion of the stomach, brain, or kidneys, producing the vomiting, yet there have now been a sufficient number of cases recorded to prove that pure functional vomiting may lead to death usually by exhaustion, sometimes by hæmatemesis brought on by straining.

In the treatment of the condition it is noticeable that remedies directed towards the nervous irritability, such as bromide, arsenic, and morphia hypodermically, have a better effect than drugs acting as simple gastric sedatives such as bismuth, iodine, ipecacuanha, or opium by the mouth. In the present case, for instance, the first attack of vomiting was immediately checked by putting the patient on large doses of bromide by the rectum, although many drugs had been previously given without effect. It is noticeable that rectal feeding which so quickly checks the vomiting of gastric ulcer, is frequently useless in these cases. Many of these patients do extremely well on Weir

Mitchell's system of rest, isolation, massage, and frequent feeding. Other severe cases are much relieved by feeding with the stomach-tube or by frequent washing out the stomach; but unfortunately the impossibility of excluding gastric ulcer will often prevent the use of a stomach-tube as in the present case. It must be remembered that in many cases the vomiting is directly brought on by gastralgia, or neuralgic pain of the stomach, and whatever will prevent the pain will cure the vomiting; and it is in this way that use of the constant current recommended by Leube, one pole being applied to the painful point and the other over the spine behind, may be found serviceable.

Renal Hæmaturia without known Lesions.

—M. L. Harris closes an article on the above subject with the following statements:—(1) There is a condition of renal hæmaturia not due to the usually accepted causes, namely, acute nephritis, calculi, tuberculosis, septic infection, malignant and non-malignant new formations, hæmophilia, injuries, malaria, intoxications, &c. (2) There is probably in these cases a local lesion in the kidney which may be strongly influenced by the nervous system. (3) With our present knowledge we are unable to state what the pathologic changes are. (4) These cases have not been benefited by the usual hæmostatic remedies. (5) After a reasonable trial of other methods of treatment, including tonics, cold baths, &c., if unsuccessful, simple nephrotomy should be performed. (6) Owing to the almost uniform success of simple nephrotomy primary nephrectomy should never be performed.

Medicine, June, 1898.

Sphincteric hysterotomy is indicated in cases in which it is necessary to insure evacuation of the contents of the uterine cavity, to facilitate involution of the uterus, and to prevent the upward extension of any infective process toward the oviducts. It is regarded as a measure of radical treatment in cases of metritis and of affections of the uterus complicated by septic inflammation, particularly uterine flexions and dysmenorrhœa of uterine origin, and also advanced retraction of the cervix. It acts by allowing complete evacuation of the uterine cavity, and by facilitating involution of the diseased organ.—Sajous' *Monthly Encyclopedia*.

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A CLINICAL LECTURE

ON A CASE OF

POPLITEAL ANEURYSM TREATED BY EXCISION OF THE SAC.

Delivered at the Central London Sick Asylum,
March 3rd, 1898, by

A. PEARCE GOULD, M.S., F.R.C.S.,

Surgeon to Middlesex Hospital.

GENTLEMEN,—One needs no excuse for selecting for our subject to-night a case of popliteal aneurysm. Aneurysm has interested surgeons for many centuries, and some of the greatest names in the history of surgery are indelibly associated with the question of the treatment of aneurysm. From whatever standpoint we regard the subject—historical, pathological, physiological, or clinical—it is full of interest. But there are two special reasons which have led me to choose this subject. One is that we are just now witnessing one of those curious changes which have led careless observers to consider that we are as fickle in our therapeutics as ladies are in their dress. Let me just read to you a paragraph from the eighth edition of Erichsen's 'Surgery,' in which reference is made to the "old" operation for the application of a ligature to the artery on each side of an aneurysm: "This operation, as performed by any of the older surgeons on any of the larger arteries, as the popliteal, was not only so difficult in itself that surgeons were seldom willing to undertake it, but was so fatal in its results, being commonly attended by secondary hæmorrhage in consequence of the artery being ligatured in a diseased part, or by diffuse inflammation, suppuration, and gangrene in the deeper tissues of the limb operated upon, that recovery after its performance was considered a marvel, and most surgeons preferred submitting the patient to amputation at once. At the present time, however, the difficulties are much diminished by the bloodless method of operating, and the

subsequent dangers by the improved methods of treating wounds. The operation may, therefore, be undertaken with less hesitation as a last chance of saving a limb in which an aneurysm has become diffused."

The reader of this passage would naturally conclude that this "old" operation had been entirely and for ever superseded by other methods of treatment, and that it found little, if, indeed, any place in the surgical practice of to-day.

And yet it is to that very operation that I wish to draw your attention this evening—this operation so difficult, so dangerous that the boldest surgeons feared to practise it, and the most successful operators rarely performed it with success—because it seems that surgeons are likely to revert to it in the treatment of many cases of aneurysm. Hunter's name and reputation by no means rest solely upon the great work which he did in the introduction of the proximal ligature in the treatment of aneurysm, but when his great name is mentioned we at once think of this as one of his chief claims to our gratitude. And rightly so; for the improvement in the treatment of aneurysm which his method of ligature of the artery far above the sac effected was enormous, and it seemed that the old operation was dead and never to have a resurrection. Yet within the last few years that "old" operation has been revived, and it promises to be the operation of the future.

The other reason for my choice is that I have recently had under my care in the Middlesex Hospital a case in which I practised this operation, a few months after the Hunterian operation had been successfully performed by another surgeon, for an aneurysm on the opposite side. It therefore affords a very good opportunity of comparing the two operations, and one that I feel bound to avail myself of. The history of the case is as follows:—The patient was a soldier aged 28, the son of healthy parents, with nothing of importance in his family history. His personal history was more interesting. When he was a boy of fourteen years of age he was operated on for varicose veins in both legs and for varicocele. Some would be inclined to say that the occurrence of varicose veins of both legs and in the spermatic plexus in a boy of fourteen showed a special tendency to weakness in the vein walls, and that the aneurysms which afterwards developed were the result of a similar condition in

the popliteal arteries. In India the man had an illness which, he was told, was syphilis, but in my opinion it is exceedingly doubtful whether the man really had syphilis; he has certainly had no sign of the disease since then. In May, 1897, he noticed pain in the right knee, and he was sent into the military hospital, where an aneurysm was discovered in the right popliteal space. In July, 1897, the army surgeon, Mr. Hudlestone, ligatured the superficial femoral artery in Scarpa's triangle. The case did well, the wound healed, and the aneurysm consolidated, but the man had to be kept in bed for several weeks, and remained in the hospital until November owing to contraction of the right hamstring muscles. Very soon after the operation on the right side Mr. Hudlestone noticed a small aneurysm in the left ham. While he was lying in bed this did not increase in size, but when he began to walk about it steadily grew larger, and in January of this year the patient was invalided out of the army and admitted to the Middlesex Hospital under my care.

One other fact in his history is important; he told us that for a year previous to his first operation he had been exercising much in the gymnasium and had often struck the back of his ham on the horizontal bar, and by hanging by his knees, and in other ways, had possibly injured this part. This fact had probably an important bearing upon the ætiology of these aneurysms.

When admitted he was well nourished and apparently in good health; he had a certain amount of stiffness in the right knee, which he could not quite extend. The muscles of the right leg were flabby and the limb was cold. I could not detect any pulsation in either tibial artery at the ankle-joint. There was a sound scar marking the site of Mr. Hudlestone's operation, and in the ham there was a slight pulseless thickening in the site of the cured popliteal aneurysm. In the left ham he had a considerable swelling which had all the characters of a globular popliteal aneurysm, about the size of an orange. The limb could not be bent beyond a right angle, owing to the size of the swelling in the ham; it could not be extended to within an angle of 30° of a straight line, and there was a certain amount of constant pain at the back of the knee. One could feel the pulsation in the tibial arteries at the ankle; there was neither anaesthesia, oedema, nor enlarged veins, nothing to show embarrassment

of the venous circulation. The heart was healthy, the urine normal, and the arteries elsewhere were soft and supple, showing no evidence of disease. Except for this popliteal aneurysm, therefore, on the left side, and the cured popliteal aneurysm on the right side, he was a healthy, robust man.

I determined to treat this second aneurysm by the method of double ligature or excision, and this was done on the 8th of January. After the patient was anæsthetised, an Esmarch bandage was wound up the limb to the groin, and then Petit's tourniquet was placed round the limb at the groin, and the bandage was removed. The patient was turned on to his face, and an incision six inches long was made over the ham. After dividing the popliteal fascia I found the internal popliteal nerve; this I separated from the sac and held aside. Close over and adherent to the sac of the aneurysm was the large popliteal vein. That I separated, and followed up to the highest part of the popliteal space where I exposed the artery above the aneurysmal sac, and put a sterilised silk ligature around it. I then followed the sac downwards, separating the vein as described, and put a ligature around the popliteal artery at its lowest part, close above the bifurcation. I next opened the sac, turned out a quantity of clot, and cut away a considerable portion of the sac wall itself. Up to this time there had been no hæmorrhage, but on loosening the tourniquet there was a free gush of arterial blood from a branch opening into the sac of the aneurysm on the outer side, close beneath the popliteal vein. There was some little trouble in securing this artery, but it was eventually tied, and after that the wound was dry. It was closed, a dressing was put on lightly, and the limb was wrapped up in a thick layer of cotton wool, and the man was carried back to bed, where the limb was slightly raised and surrounded with hot bottles. I need not trouble you with the details of the convalescence. The wound was dressed next day because it was feared that the dressing was a little tight. On the 21st he was again dressed, and the wound was found healed; the stitches were removed, and a collodion dressing was put over the scar. The circulation in the limb had been satisfactory from the first, and had not occasioned any anxiety. The man experienced hardly any numbness or tingling in the foot. On the thirteenth day after the operation we felt distinct pulsation in the tibial arteries at

the ankle. On January 23rd he got up for the first time, and he left the hospital on February 3rd, the operation having been done on January 8th. He came back to see me three weeks after that, when he was able to walk thoroughly well; he had practically full movement in the limb, and had no pain or trouble of any sort.

There are many points of interest in this case, particularly the ætiology of the popliteal aneurysms, and the occurrence in an otherwise healthy man of two aneurysms at exactly corresponding points of the arterial system. But I wish to-day to consider the case chiefly from the point of view of treatment, for it presents a very good opportunity of comparing the Hunterian operation with excision of the sac. As a rule, we have to draw our comparisons between different modes of surgical treatment from the results obtained in different patients, and often, too, by different surgeons. The opportunities of comparing methods of treatment—particularly operations—in the same patient, and at a short interval of time, are not frequent, and it is because the two operations I wish to compare were carried out within a period of six months in this man under circumstances as closely similar as can be desired, that this case is specially valuable for our purpose. The two operations may be compared in four particulars:—(1) The ease or difficulty of the operation; (2) the mode of cure; (3) the interference with the circulation in the limb it causes; (4) its range of applicability. The patient was an intelligent man, and his evidence on the relative value of the operations was not to be disregarded. When we asked him whether he experienced any difference in the results of the two operations, he was very emphatic in his preference for the operation of excision. In estimating the value of this testimony we must remember that when one asks a patient to compare one's own treatment with another treatment that has been previously followed, his bias is to speak favourably of our own treatment rather than of that of another surgeon. But we cautioned him and tried to eliminate this source of error, and still he was perfectly clear that he had much less numbness and much less discomfort from imperfect circulation in the limb after the excision of the aneurysm than after the ligature of the femoral artery. Our observations of the case fully bore out this statement, for when we examined the right limb six months after the

ligature of the femoral artery, the circulation in it was still a little imperfect, and no pulse was to be felt in either tibial artery at the ankle, and he had a little tingling and numbness at times, whereas when he left to go to a convalescent home, within a month of the second operation, he had no tingling or numbness in the leg, the circulation in the limb was quite as good or rather better than in the other limb, and the tibial pulse could be felt. The man was very emphatic that two days after the second operation he had very much less discomfort in the limb than after the ligature.

Of far more value than his statement, however, is the fact we all observed—that, while even eight months after the first operation no pulsation could be detected in the tibial arteries at the ankle joint, six weeks after the second operation we could plainly feel the pulsation in the posterior tibial artery on the side on which the aneurysm had been excised. This was positive objective evidence that the circulation had not been so grievously interfered with on the left side as on the right side.

Let us now take up our four points of comparison in order:

1. *The operation itself.*—To put a ligature around the superficial femoral artery is a very much easier and simpler operation than to excise a popliteal aneurysm. Having done both, one can speak very definitely about that. I would certainly not advise any one who is not familiar with operating, and who has not all the conditions that we have in hospitals, to operate by excision; I would urge him rather to adopt the method of ligature above the sac. It is no exaggeration to say that the operative difficulties of the most troublesome case of ligature of the superficial femoral artery are less than those of the simplest case of excision of the sac of a popliteal artery. This strong statement would not apply, however, to cases of aneurysms in all situations, but I think the general statement is true that the Hunterian operation is easier to perform than the "old" operation of Antyllus. This difference is not to be ignored. There are many cases in which a short, simple operation can be safely borne where a long complicated operation becomes at once a dangerous procedure. In all cases simplicity in an operation is a distinct element of safety.

2. *The mode of cure.*—We must never forget that

the essential step in the cure of an aneurysm is the permanent obliteration of the artery from which it springs. It is not enough for the sac of the aneurysm to fill with clot; the artery itself must become occluded; this is the "irreducible minimum." Hence it follows that some aneurysms are absolutely incurable—*e. g.* those of the ascending aorta—for the process of cure of the disease would itself prove fatal. Now, the operation of Antyllus aims at securing just this "irreducible minimum." It permanently occludes the diseased artery, and if the operation is itself successful the cure of the aneurysm is certain. It is the most direct of all methods of treating aneurysm. The Hunterian operation, on the other hand, aims at curing the aneurysm by indirect means, by so lessening the tension in the sac as to induce coagulation of the blood within it, and by the extension of this thrombus into the artery, when by its organisation it effects the permanent obliteration of the vessel, on which the cure depends. You see, therefore, that the cure of the aneurysm does not depend directly upon the success of the operation itself, but indirectly upon the changes in the circulation that operation effects. The ligature of the artery may be successfully performed, the artery being obliterated, and the wound healing without any complication, and yet the aneurysm may be unaffected. Many such cases occur, and although the percentage of success after the Hunterian operation is high, yet failure to cure is not very infrequent; and not being dependent upon any details of the operation itself, its success is out of the power of the surgeon himself to command. We may, however, say this: The more nearly the Hunterian operation approaches that of Antyllus, the more certain is its therapeutic effect. In other words, the nearer the ligature is placed to the sac of the aneurysm the greater the prospects of attaining obliteration of the diseased portion of the artery, and therefore the cure of the aneurysm. At the risk of repetition I must again insist upon the fact that an aneurysm is cured not by the formation of a clot within it, nor by any change in its sac or contents, but by the permanent occlusion of the artery from which it springs. Inasmuch as the "old" operation directly and certainly accomplishes this end when successful as a mere operation, it is superior as a method of treatment to the Hunterian operation, which at best only indirectly cures the

aneurysm, and may fail in this even when most successful as a mere operation.

3. *The interference with the circulation in the limb.*—By the operation of excision the main artery is obliterated at the seat of the aneurysm only—an inevitable accompaniment of cure of the disease—and the circulation in the parts beyond is dependent upon anastomosing channels from the artery above this point to the arteries below it. This is, let me repeat, the “irreducible minimum” of interference with the arterial circulation that the cure of an aneurysm by any means must involve. By the operation the sac is either removed or emptied, and the tension of the parts is thereby lessened. This, of course, favours the development of the anastomotic circulation around the sac.

The Hunterian method in many cases interferes with the circulation of the limb to a greater extent. Take the case of our patient. When the superficial femoral artery was tied, a clot formed in the vessel which we may be reasonably certain extended up as high as the mouth of the profunda artery and an uncertain distance down the femoral artery; that clot became vascularised and organised, and the artery was converted into a fibro-cellular cord. The blood then flowed into the limb through the profunda artery and the other vessels above, and by various anastomosing channels it reached the limb below, and a trickling stream of blood passed through the aneurysmal artery and into the limb below.

All went well, the heart was strong, and the arteries were not the seat of atheromatous or other disease which hardened them, so that these anastomosing vessels dilated and carried a sufficient stream of blood into the limb below. But it was a slow trickling stream, which had lost the full force of the heart: the blood pressure was quite low, and under such circumstances a clot formed in the aneurysm in the ham either gradually or suddenly. The clot then extended through the mouth of the aneurysm into the artery, and grew in the artery until it blocked it up. The result of the first operation upon this patient's arteries was by the ligature itself to place a permanent block in the superficial femoral artery at the upper part of the thigh, and then, by the cure of the aneurysm, to place a second block in his main arterial system in the popliteal space. The point is that in the Hunterian operation there is and must be an

obliteration of the artery at the seat of the ligature, and an obliteration of the artery at the seat of the aneurysm. In some cases there is one continuous block extending from the ligature to beyond the aneurysm, as in the operation for carotid aneurysm, and in any case where the ligature is placed close on the proximal side of the aneurysm. But in the case we are considering—ligature of the femoral artery at the apex of Scarpa's triangle for popliteal aneurysm—this only occasionally happens. Savory found that of seventeen cases examined by him, in fourteen the femoral artery was patent between the ligature and the aneurysm. It follows from this that the limb below the aneurysm is dependent for its blood supply either upon a double series of anastomosing vessels—one passing from above to below the ligature, and the other from below the ligature to below the aneurysm—or upon a very long series of anastomosing vessels passing from the profunda artery above into the tibial arteries below.

We must not forget that there are two special hindrances to the free development of the second or distal series of anastomosing vessels. One is the low blood-pressure in the arteries beyond the ligature, and the other is the presence of the aneurysm, which, if large and full of clot, may exert considerable pressure upon the muscles and fasciæ in which the anastomosing vessels run.

[Mr. Pearce Gould then enumerated the vessels which would carry on the circulation in the case of ligature of the superficial femoral artery for popliteal aneurysm, and also those which would enlarge in case of excision of the aneurysm.]

It is this fact that explains the frequency with which gangrene has been met with after the Hunterian operation, and the frequency with which gangrene follows ligature of the artery for aneurysm as compared with ligature of an artery for any other condition. For instance, if a man stabbed in the apex of Scarpa's triangle, has the artery ligatured simply for the treatment of the wound of the vessel, unless he has lost very much blood, or is the subject of some visceral disease, or his vessels are very atheromatous and rigid, the operation is not attended with serious danger of gangrene. But whenever a man has his femoral artery tied for popliteal aneurysm there is danger of gangrene. That this is no mere theoretical point is well illustrated by what we observed in the

patient whose case is the text of this lecture. He himself was very conscious of the marked difference in the circulation in the leg and foot after the two operations, and we were able to corroborate most fully his observation. As I have already pointed out, this case forms the best possible means of comparing the two methods of treatment in this particular, for the other conditions are identical, as they never can be when the comparison is made between the results in different patients. You cannot so well compare two operations in two different patients as two operations in the same individual; in the two patients you may have a different strength of heart, and a different condition of arteries; but in our patient we had the same condition of heart and arteries, and we may fairly assume that all the physiological conditions were identical when the two operations were performed. After one operation the patient had pulsation in the arteries at the ankle-joint some months quicker than after the other, showing that the anastomotic circulation was very much better in the second case than in the first, and the capillary circulation was correspondingly more active. This is one strong ground upon which the operation of Antyllus rests. It removes or cures the aneurysm with the minimum of obstruction to the arterial circulation.

4. *Range of applicability.*—Upon this point I would make only two remarks. And the first is that excision of the sac of an aneurysm can sometimes be practised successfully where a ligature far above on the cardiac side of the sac is either impossible or attended with grave difficulty and danger—such a case is an aneurysm of the subclavian artery. The other remark is that this operation can be employed in cases of leaking and ruptured aneurysms where the Hunterian operation is inapplicable.

If we ligature the artery above in a case of diffuse aneurysm we court failure. In the case of a ruptured sac it is certain that the limb below will become gangrenous, and if we do not lose our patient we shall have to amputate his limb to save his life. The operation of excision of the sac or double ligature can be and has been carried out successfully in cases of diffuse aneurysm; and whatever may be its scope in circumscribed uncomplicated idiopathic aneurysm, it is the operation which ought to be practised in all cases of leaking rupture of an aneurysm, and that without any

hesitation or delay. The surgeon should put on a tourniquet, make a very free incision, turn out all the clot and fluid blood that is in the tissues and lying in the sac, find the mouth of the vessel, and tie it both above and below the sac. This operation for diffuse aneurysm is no doubt of immense advantage.

To sum up, then, the results of our comparison of the two operations, we have found that the operation of Antyllus is more difficult of execution than the Hunterian operation (with a few exceptions); it cures the aneurysm directly, and with the minimum of interference with the circulation in the parts beyond, and it can be successfully employed in certain cases where the Hunterian operation cannot. Hunter's operation has the one great advantage of being in most cases the easier operation to perform.

There are two ways in which the operation may be done, namely, first by exposing the sac, and trying to separate all the parts around the sac from it, especially the neighbouring vein and the nerve or nerves, and then ligaturing the artery above and below the mouth of the aneurysm, and excising the mass, that is the aneurysmal sac. This is a difficult operation, and sometimes a very tedious one. Nerves, and still more often veins, are liable to be firmly blended with the sac of an aneurysm, and to injure either is a serious accident. It is more dangerous and serious to wound a vein than to wound a nerve in these cases, because we are going to take off the *vis a tergo* of the heart's action from the circulation, and if we put an obstruction on the venous side by a wound, and then a ligature of a vein, the risk of gangrene will be very greatly intensified. So that that method of operating is both tedious and dangerous. The other method is, having exposed the sac of the aneurysm, and having identified the position of the vein as the part most likely to be blended with the sac, and most liable to be accidentally injured, to make a free incision into the sac of the aneurysm, turn out all the clot, find the mouth of the vessel where it opens into the aneurysm, into which a probe or director may be introduced to facilitate the cleansing of the artery just above and below the mouth of the aneurysm, and put on the ligatures. Any other opening into the sac or branches arising from the artery there must be carefully ligatured, and then the wound washed

out and closed. That is to say, we tie the artery above and below the aneurysm after a free incision into the sac, and turning out all the clot, without troubling to excise the aneurysm itself. Experience has shown that this mode of operating is simpler, quicker, safer, and just as efficient as the first. It is a tempting thing to dissect out an aneurysm and preserve it in a bottle for exhibition purposes, but the desire for that should not be gratified at the expense of safety. It is a difficult operation to dissect out an aneurysmal sac of any size. It forms strong adhesions, and veins, nerves, and muscles get displaced from their normal position, and in dissecting out the sac, one is very apt indeed to wound them. Moreover, there is no compensating advantage for all the surgeon's time and trouble, and for the danger to which he exposes the patient in excising the sac.

This operation has long been practised for traumatic aneurysm. But in aneurysm from disease it has been urged that the vessel close to the aneurysm, where the surgeon puts his ligature, is so likely to be atheromatous, or in an otherwise morbid state, that the ligature will not hold, and the repair of the artery will not take place soundly, and that secondary hæmorrhage will occur. More exact pathological knowledge has shown that it is a mistake to think that the artery close to the mouth of an aneurysm is so very frequently diseased. It is not right to say that you are much more likely to find the upper part of the popliteal artery diseased in cases of popliteal aneurysm than the highest part of the superficial femoral artery. Then, with the introduction of the antiseptic and aseptic treatment of wounds, especially the use of sterilised ligatures, the risk of secondary hæmorrhage after ligature has been enormously diminished, so that the surgeon does not really fear secondary hæmorrhage in these cases. So far as the comparison between diseased arteries and injured arteries is concerned, I do not think there is any reason why the operation which has been shown to be so successful in traumatic aneurysm should not be practised in idiopathic aneurysms.

Of late years this operation has been very extensively resorted to. In this country Mr. Littlewood, of Leeds, has probably had more experience of it than anybody else, and he has obtained excellent results. He has published

several cases. Not only have popliteal aneurysms been excised, but a subclavian aneurysm has been successfully treated in the same way. On the continent, especially in France, the operation is now frequently practised, and it is coming to be looked upon as *the* routine or proper one for aneurysm—a change of ideas and of practice that would have been deemed incredible by surgeons twenty or thirty years ago. But it is not a mere swing of the pendulum or a change in surgical fashion; it is the result of greater knowledge and the general advance in surgery; our knowledge that the artery near an aneurysm is not necessarily diseased or specially prone to be diseased, and our greatly improved technique. Esmarch's bandage renders the operation bloodless, anæsthesia keeps the patient motionless during operation, and the antiseptic and aseptic treatment of the wound with the use of sterilised ligatures properly applied has abolished secondary hæmorrhage.

"Anæsthetics and Counter-irritants."

To the Editor of the CLINICAL JOURNAL.

SIR,—Apropos to recent discussions in your columns will you allow me to remark that if patients are smothered with ether that it is the most rapid anæsthetic we possess; indeed, I have often operated upon five children within twenty minutes by its use.

For more prolonged operations I have found that ether nebulised with nitrous oxide in Oppenheimer's inhaler gives the most satisfactory results.

As to the effect that the incidence of one disease has upon the progress of another, I was surprised that none of the speakers noticed that an attack of smallpox will frequently arrest or cure phthisis.

Yours faithfully,

CHARLES BELL TAYLOR, M.D.,
*Surgeon Nottingham and Midland
Eye Infirmary.*

Pemphigus Chronica Vulgaris.—In a boy of five years the disease appeared after scarlet fever one year before. During the year the child was not free from the eruption. No bacteria could be demonstrated. Arsenic, white precipitate ointment, and baths of potassium hypomanganate constituted the treatment.

American Journal of Obstetrics, June, 1898.

A CLINICAL LECTURE ON CAUSES OF HYPERTROPHY OF THE HEART.

Delivered at the Hospital for Consumption and Diseases
of the Chest, Brompton, July 6th, 1898,

By **SIDNEY MARTIN, B.Sc.Lond., M.D., B.S.,
F.R.S., F.R.C.P.,**

Assistant Physician to the Hospital and to University
College Hospital.

GENTLEMEN,—Before I ask you to examine the patients I propose to make a few remarks on the causes of hypertrophy of the heart. The heart hypertrophies when it has more work to do. This increase of work has often extended over months, or perhaps years, the hypertrophy being, in the majority of instances, a slow and not an acute process. As regards the power of hearts to hypertrophy, that depends partly on the diseased condition which, so to speak, initiates the hypertrophy, and also partly on whether the heart is that of a young adult or that of an older person, because the greatest forms of hypertrophy are seen in the youngest hearts; and the same degree of disease which would readily produce hypertrophy in a young heart will not excite hypertrophy in the old heart, which, in many instances, seems incapable of hypertrophy.

The causes of hypertrophy may be divided into two classes. In the first you have causes in the heart itself. The general cause in the heart itself is an obstruction to the flow of blood through the heart. I will explain that in detail presently. The first effect of obstruction to the flow of blood from one cavity of the heart to another, or from the heart into one of the great blood-vessels, or to the flow of blood back from one cavity into the cavity it has just left, is dilatation. That is, one or other cavity of the heart tends to become too full, and this cavity becoming too full tends to dilate, and hypertrophy is the effort which the heart makes to overcome the dilatation.

Another class of causes of hypertrophy of the heart are changes in the peripheral circulation—that is to say, the circulation in the arteries and arterioles. That is the second great cause of

hypertrophy, such hypertrophy affecting only the left ventricle. Hypertrophy of the left ventricle ensues when there is chronic disease of the small arteries, such as occurs in many cases of chronic renal disease. It is necessary to state that in the majority of instances a simple degeneration of the arteries irregularly placed, such as occurs commonly in atheroma (brittle arteries), does not lead to hypertrophy of the left ventricle, inasmuch as there is no actual hindrance, no great resistance to the circulation of the blood. But when you have a large number of small arteries, or arterioles as they are called, with a diminished lumen, then you have an enormous resistance offered to the flow of blood from the heart through the arteries to the capillaries and so to the veins, and you get an increased arterial pressure. That increased arterial pressure leads eventually to hypertrophy of the left ventricle of the heart.

I have made a few diagrams on the board which illustrate some of these points a little more clearly perhaps than a simple description in words would do. One of the simplest examples of hypertrophy of the heart is afforded by aortic obstruction. I have drawn two thick lines to show that the aortic valves are stenosed—that is to say, they are stiff, contracted, joined together, so that the blood has difficulty in passing from the left ventricle into the aorta. The result of that obstruction is very simple, namely, that the heart hypertrophies to overcome it. That it successfully does so is shown by the fact that many patients for a number of years are able, by the hypertrophy of their left ventricle, to keep up the circulation sufficiently vigorously to maintain the needs of their tissues. That is the early stage, and a chronic stage of aortic stenosis, in which there are no symptoms, frequently happens. The later stage of aortic stenosis may be represented in the next diagram, where you still have the hypertrophy of the left ventricle, which contracts vigorously, but the stenosis being so great the hypertrophied left ventricle is not strong enough to send sufficient blood through the narrow orifice, so that the previously hypertrophied ventricle gets more dilated. The result of that is that the mitral valve becomes incompetent, you get dilatation of the left auricle, dilatation of the right ventricle, and so of the whole right side of the heart. In some of these cases, therefore, dilatation of the right side of the

heart is the final stage of aortic obstruction. Aortic stenosis is a simple condition because you have got obstruction to the flow of blood, and you can easily imagine why hypertrophy takes place behind it. In the case of aortic regurgitation, what really happens is that when the left ventricle contracts, a certain amount of blood is sent into the aorta through the open valves, but during the diastole, or just immediately after the systole, the blood regurgitates into the ventricle, and at that same time the blood is flowing from the auricle through the mitral orifice into the ventricle, so that the ventricle gets overfull of blood. The left ventricle tends to become overfull, to be distended, and so the ventricle acts more vigorously to expel the contents, which are greater than normal. Of course, as is well known, the final stage of chronic simple aortic regurgitation is associated with dilatation of the mitral orifice and enlargement of the right side of the heart. But many cases exist for a number of years in the condition described, the hypertrophy of the left ventricle being sufficient to keep up the needs of the tissues by supplying them with blood.

The explanation of the hypertrophy in mitral regurgitation, which occurs with both right and left ventricles, is arrived at by simply carrying the explanation, so to speak, a cavity backwards. Instead of regurgitation occurring into the left ventricle, as it does in aortic regurgitation, it occurs into the left auricle, and therefore the first increase of pressure, the first dilatation, occurs in the left auricle itself. That increases pressure in the pulmonary system; and the consequence is that the right ventricle, finding a difficulty in propelling blood from the right side of the heart through the lungs to the left, hypertrophies.

The reason the left ventricle hypertrophies in mitral regurgitation is, that with this widely patent mitral orifice the whole of the left side, not only the left auricle to which extra blood is shot from the left ventricle at the systole—not only does that become more full of blood, but the whole left side tends to become overfull, and instead of acting as two cavities they act as one, the mitral valve never shutting.

In mitral stenosis there is another condition of things. There are two conditions in which mitral stenosis occurs, one of which offers some difficulty in explanation. The drawing here may be con-

sidered to represent a classical mitral stenosis. Here is the button-hole mitral represented by the narrow orifice; sometimes it is funnel-shaped, with shortened chordæ tendineæ and shortened and thickened musculi papillares. For the blood to be sent from the left auricle into the left ventricle great increase of pressure is required, and there is an increase in the left auricle. The blood reacts backwards through the pulmonary veins to the right ventricle, and it is that increase of pressure in the pulmonary arteries owing to the back pressure from the auricle that causes the hypertrophy of the right ventricle. And, as is seen clinically in many cases, that condition of things lasts for many years without anything further happening, the right ventricle being sufficiently hypertrophied—sufficiently strong—to keep up the pulmonary circulation and to supply the left auricle, and so also the left ventricle, with sufficient blood to carry on the circulation.

But there is another class of case in which mitral stenosis exists, and is diagnosed during life, but one finds that at death, on an examination of the body, the left ventricle, instead of being small and not hypertrophied, is hypertrophied. There may be therefore a button-hole mitral with hypertrophy of both sides of the heart, such as occurs in mitral regurgitation. The difficulty, of course, is to explain how it is that the left ventricle hypertrophies, because, as is evident, there is a great difficulty in the blood getting from the left auricle into the left ventricle, and therefore one cannot imagine that there is any increase of pressure in the left ventricle with a button-hole mitral. It seems to me that the only explanation of such cases is that the hypertrophy of the left ventricle has existed before the stenosis. One knows that in these cases of mitral stenosis that the damage which is first done to the valve is one of destruction, leading to regurgitation; and the regurgitation would, in the ordinary course of events, lead to hypertrophy of the left ventricle. Subsequently by the organisation of the adhesions on the edges of the valve the valve becomes stenosed and contracted, and you have a stenosed valve with hypertrophy of the left ventricle. I must say that in the majority of cases at death one does not find an atrophied left ventricle. That is a point which is mentioned in books, but it is not a fact. I believe that in the majority of cases post mortem one finds the left

ventricle is not atrophied in mitral stenosis. It is perhaps small, or it may be dilated, or it may be hypertrophied, but it certainly is not atrophied.

I have put another diagram here to show the condition of things which obtains in some cases of congenital cardiac disease. The commonest form of congenital cardiac disease is stenosis of the pulmonary artery, which is due to a malformation and not to an inflammatory condition. The stenosis consists in kinking or narrowing of the artery just above or at the level of the cusps. The second condition is that the cusps, instead of being semi-lunar in shape are very small, and one of them may be absent. The result of that stenosis is a simple hypertrophy of the right ventricle without any hypertrophy of the left. But it is not at all uncommon for another condition to be associated with the pulmonary stenosis, and that is a communication between the two ventricles by a gap in the septum at the base, in the way I show you. Undoubtedly the great obstruction on the right side of the heart due to the stenosis of the pulmonary artery is relieved by this communication between the two ventricles. I must say that I do not think it is really an explanation of it, because if you examine this orifice between the two ventricles carefully you will observe that it is funnel-shaped, the broader part of the funnel being in the left ventricle, so that from that shape it appears that no blood would pass from the right ventricle to the left. It looks so from the shape of the orifice. On the other hand, one must remember that with this stenosis, which is the first thing to happen, and the hypertrophy of the right ventricle, there is a greatly increased pressure on the right side of the heart, which may be greater than on the left side, where there is no hypertrophy, and so you may get blood passing from the right ventricle into the left.

As regards the clinical signs of hypertrophy, in many instances, of course, the diagnosis of hypertrophied right or left ventricle is simple. That is, as regards the left ventricle, the heart's apex-beat, which normally is at the fifth space, from three to three and a half inches from the mid-sternal line, is carried downwards four to four and a half inches or downwards into the sixth space, or outwards and downwards. The fallacies as regards this diagnosis may be that the heart is pushed over, or that it is dragged over, a fallacy which can be obviated by recognition of the diseased con-

dition of the lungs and pleura. The occurrence of displacement of the cardiac beat outwards and downwards, and a heaving impulse is the point which enables hypertrophy of the left ventricle to be diagnosed. But, of course, there are not a few cases, especially in middle age, in which the heaving impulse is absent, owing to the heart being covered by the lung. In these cases the diagnosis of hypertrophy, which depends chiefly on the character of the impulse, is difficult, if not impossible; it is then purely a matter of inference from the size of the heart. As regards the right side, the question of epigastric pulsation often gives rise to a great deal of difficulty in the diagnosis of whether the heart is hypertrophied or not. There is no doubt whatever on examination of a large number of chests, and many people have come to the same conclusion, that in some people epigastric pulsation is not a sign of disease; it cannot be taken to mean hypertrophy of the right side of the heart in every instance. If, however, with this epigastric pulsation you have a very forcible heaving impulse, that means hypertrophy. A mistake which I have often seen made in this connection, and it may be easily made by anybody, is that the impulse in the epigastrium may be forcible, but may be due to the left ventricle, which is either dragged or pushed over to the right side, dragged over by adhesions in the right, or pushed over by effusion in the left pleura. And especially, as frequently observed in congenital cardiac disease, it is very difficult, if not impossible, to diagnose hypertrophy of the right side of the heart, owing to the narrowness of the costal angle. In people in whom the costal angle is very narrow it is impossible to feel the pulsation of the right ventricle, and it is therefore impossible to be certain whether it is heaving or not. One not infrequently finds post-mortem in cases of congenital cardiac disease that the hypertrophy of the right heart has not been diagnosed during life.

Some stress has been laid on the character of the second sound in the diagnosis of hypertrophy of the left or right side of the heart; and in cases of hypertrophy of the left ventricle you may get accentuated aortic second sound. This of course does not occur in valvular disease of the aortic valve; it occurs only in hypertrophy of the left ventricle which is due to renal disease, and is frequently absent or difficult to diagnose. In hypertrophy of the right side of the heart, not due to

disease of the pulmonary valves, the accentuation of the pulmonary second sound is a better sign in the diagnosis of the condition of hypertrophy than it is in the left side of the heart.

But there are cases in which you have an hypertrophy of the left ventricle ; you have an epigastric pulsation and you have no murmur in the heart. One supposes you have a condition of things like the heart of mitral regurgitation. The diagnosis rests between mitral disease and chronic renal disease associated with emphysema. The points to pay attention to under such conditions are these : you must remember that the murmur in mitral regurgitation and in mitral stenosis may be absent at the time of examination, although it may be detected afterwards from time to time. In mitral stenosis, which forms a large number of such cases, the second sound is frequently accentuated at the pulmonary cartilage, and that is one of the points in which it differs from renal disease, where the accentuation takes place at the aortic valves, and is associated with a pulse of high arterial pressure. A much more important sign in the diagnosis of these cases where one cannot tell whether it is renal or mitral disease, from the condition of the heart alone, is to take from time to time the relation between the heart-rate and the pulse-rate. There are but few cases of mitral stenosis—and I should say there are no cases of well-marked mitral stenosis—which on excitement or exertion do not show that many of the beats of the heart do not reach the wrist. And if there is a large disproportion between the rate of the heart as felt at the wrist, and the rate of the cardiac beat, the condition is in all probability one of mitral stenosis, and not of renal disease. There are remarkable cases in which you have mitral disease associated with renal disease and a pulse of high arterial pressure ; mitral disease not excessive in amount of course, but quite distinct enough to give murmurs, and hypertrophy of the heart and increased arterial pressure.

[A number of patients illustrating the various cardiac conditions were then demonstrated by Dr. Martin and examined by the class.]

THE OPERATIVE TREATMENT OF CANCER OF THE BREAST.

Abstract of an Address delivered at Beckenham before
the Beckenham and Penge Medical Society, Friday,
March 11th, 1898,

By MARMADUKE SHEILD, M.B., F.R.C.S.

GENTLEMEN,—In response to the invitation of your Secretary, there are two reasons why this is a very opportune occasion for bringing the subject of the operative treatment of cancer of the breast before this meeting. Firstly, because most of you are aware the questions with regard to it have recently given rise to a debate of some magnitude, and have created some attention among surgeons in London ; and secondly, there is a reason which affects you nearer home, namely, that these cases are common in practice, and that they must therefore occur to all of you. There is probably no one in this room, in the daily course of his profession, who has not been, or will not be, called upon to advise upon the treatment of one of these cases. Therefore it is of importance that we should discuss a little in detail the history of the treatment of cancer of the breast, and especially how the matter stands at the present time.

If we go back to the ancient history of cancer of the breast—the days, for instance, of the old surgeons, Paracelsus, Antyllus, and Paulus Æginita—generally speaking the operative treatment was much the same ; cases were not operated upon until they had got to a very bad stage, that is, until the sufferers were driven to surgery by horrible pain and fungating masses of cancer on the front of the chest. The whole diseased area was swept off with a crooked knife, leaving an enormous wound. Hæmorrhage was staunched with the actual cautery or violent styptics. These methods, rough and barbarous as they were, had the one advantage of very completely and extensively removing the disease. Had such operations been done earlier, instead of waiting until the disease was so far advanced, the probability is that the results would have surpassed much of the surgery which later occurred. Following such old and rough surgery came a revulsion against what might well have been called barbarism, and sur-

geons began to operate with the idea of getting union by "first intention" of the wound which they had made. The consequence was that the operations were done very imperfectly; indeed, very frequently the cancer was not removed at all. The flaps of the wounds were shut up, enclosing infected tissue, fragments of breast and fascia, between them. The skin itself was often left infected. This is the kind of operation which was very largely done in this country fifty to seventy years ago, and in the days of Brodie, Astley Cooper, and Everard Home such operations, therefore, were not of a very complete sort, owing to the chief care being about the bringing together of the wound afterwards. Under these circumstances the term "recurrence" was a clear misnomer; future cancers in the operation area were not recurrences at all, because the disease had never been removed completely. So bad were the results fifty or seventy years ago that we find such men as Brodie, Home, and Munroe would scarcely consent to the performance of the operation at all except in some very favourable cases. In Brodie's works the suitable conditions for operation are laid down very rigidly; he only picks out a few cases where the growth is very early, very small, and very chronic,—where the skin is unaffected, the glands are not involved,—and says that only in these is it justifiable to operate. We recognise these conditions as favourable in the present day. It was precisely the same in European countries and America. Sands stated that he only operated for what he termed moral effect. Agnew declared he had never saved a single case. Sir James Paget's estimation of the results of operation on the breast, coming later, were very pessimistic. He once stated publicly that he did not believe there was one case out of five hundred which did not recur sooner or later after the operation. Kindly writing to me in 1896, he said that, speaking from recollection, he had known many people live over three years after operation, such as were now termed cures; of those living five or six years after operation he knew very few, and only one or two who lived ten years were known to him. I think the reason of all this can be largely explained by the supposition that the operations of those days were so done as to leave infected tissue behind, either in the wound or in its immediate neighbourhood.

Before we go any further it will be better for us to define the terms "complete" and "incomplete" as applied to operations for cancer of the breast. It is very difficult to explain them accurately, but by "incomplete" I should mean that the whole breast is not removed, and that outlying prolongations of mammary tissue are left behind. The mammary gland is not a round structure; we know now that the boundaries of the mammary gland are irregular, and portions of mammary tissue extend into the tissues beneath and around the breast. An "incomplete" operation would, therefore, be the removal of the breast by an oval incision, but round the opening numerous little masses of mammary tissue are left. Another factor of an "incomplete" operation would be leaving infected pectoral fascia under the breast, and yet another, would be leaving infected cancerous glands in the axilla. By the "complete" operation, on the other hand, is meant not only the free removal of the breast with all its outlying lobules and subjacent fascia, but also the axillary glands and the whole of the great pectoral muscle, the removal of the fascia over the lesser pectoral, and the dissection of tissue even from the axillary vessels.

We have now somewhat cleared the ground for the more detailed consideration of our subject. I have said that the results of operations for cancer of the breast many years ago were stated to be very bad; but when I came to inquire for myself into this, I was surprised to find, in the last twenty years, what a number of cases there were, in which extraordinarily good results had been obtained by surgeons who performed the more or less incomplete method of operating. It must be carefully remembered that these results were exceptional. Still, one found in the practice of every surgeon of long experience, some cases in which there had been long periods of freedom from disease after operation. I may mention, as notable examples, cases of Mr. Teale, of Leeds, who sent me a large number of instances of patients who remained free from disease for periods of years; also cases in which there was recurrence in the scar after long periods which was again removed, and again long freedom was observed. Mr. Croft sent me a similar series of cases; and in the debate at the Royal Medical and Chirurgical Society the other night there were few surgeons of experience who could not add to the cases I had collected some of their

own. Thus Mr. Hutchinson related two cases where patients had remained free for upwards of twenty years after the removal of the breast by the late Benjamin Phillips; Mr. Heath mentioned nine cases in his experience of long freedom, and Mr. H. Morris furnished me with a long series of remarkable cases from the records of the Middlesex Hospital, which I had not time to read. Indeed, there was abundant evidence that the results of the incomplete type of operation were not so unusually bad as they are generally believed to be, a large number of patients having long freedom from return of the disease. Again I must remind you that these are only exceptional cases collected from sources of large experience; I do not wish to quote them as an argument for insufficient operating. What we may call the first revival of complete and extensive operating in this country was a remarkable paper, which created too little attention, by Mr. Charles Moore, of the Middlesex Hospital, in 1867. The origination of the extensive operation for cancer of the breast must be placed to the credit of an English surgeon. Moore pointed out the importance of removing the outlying lobules of the breast, and described how cancer spread from a focus into the tissues round in a radiating manner. His views never took root much in this country, but they attracted attention in Germany, and in America the elder Gross took up Moore's precepts, and for many years performed a very extensive operation. So far as I could see from the observations of the large number of distinguished surgeons who spoke the other day at the Royal Medical and Chirurgical Society's meetings in London on this question, it is clear that the senior surgeons by no means advocated the complete operation in every case. Some of them said they would never clear out the axilla unless they found infected glands there, having in mind the theory of glands being protective against the invasion of cancer. It may probably seem old-fashioned for a surgeon to say now there is any protection from systemic invasion of cancer by the lymphatic glands, but it was evident that some surgeons, and those of great experience, held that view very strongly. If you introduce a dye into the skin, it becomes arrested in the nearest lymphatic gland, and from the analogy of syphilis and lymphangitis it is argued that the glands act as barriers to prevent disease getting into the system.

It has been held by surgeons of large experience that if the lymphatic glands are not infected they had better be left, and when the disease returns in the glands they can then be removed, and that the disease can thus be prevented from being disseminated through the system so quickly as otherwise would occur. To a certain extent that is borne out by some of the results of very extensive operations, for in Halstead's remarkable cases in which he removed the pectoral muscles and a large amount of tissue, a number of the cases died of visceral deposits of cancer, thus bearing out the ideas of the older surgeons. I mention that theory, not to say that I advocate it myself, but I think it right to allude to it, considering the number of very distinguished surgeons who give it support, because it appears to me to be highly suggestive and worth while remembering. Yet it has always seemed to me that if a cancerous gland be left undetected in the axilla, it simply serves as a fresh focus of disease, and the glands next above soon get contaminated, so that if a surgeon leaves the axillary glands untouched he must be very careful they are not infected.

The next point I had better bring before you is a sketch of the so-called complete operation as practised by Halstead and Meyer, founded on the pathological researches of Heidenhain.

Probably you are all familiar with the details; but, as I shall have to refer to it from time to time, it is as well to draw your attention to some of the main features of this proceeding. Time will only allow me to be brief.

Halstead's or Heidenhain's operation essentially consists in removing the breast, the infected skin, the axillary glands, the fasciæ, and the great pectoral muscle practically in one piece. An incision is made reaching from the coracoid process, sweeping round the breast and affected skin, back to the spot whence you start. The flaps are dissected back, and the breast is detached; then the incision is carried into the axilla, and the pectoralis major is divided at its origin and insertion, and the infected tissue and fascia removed in one piece; the pectoralis minor is divided, and the axillary sheath is dissected bare with a sharp knife. The latter detail is one I would not recommend except to a highly experienced operator. All large vessels are clipped before division, so that the hæmorrhage should be slight. Of course this operation takes

away a large amount of tissue and leaves a correspondingly enormous wound, and the magnitude of the operation must, in my judgment, add to the risks of it. It is true those risks can be greatly diminished by careful and rapid operating, and by strict asepsis; but still an operation of such magnitude must entail corresponding perils upon old, feeble, and bronchitic patients. We know we cannot always choose our patients for operation in cancer of the breast, and so prudence should make us select carefully what we can choose in methods of operating. For instance, think of such an operation being performed on a fat, bloated, emphysematous woman of sixty years of age; it would be extremely severe compared with the minor one of clearing out the axillary glands and fascia only. There is another objection which one is obliged to consider in reference to this operation besides the increased risk which it must entail, and that is in the movements of the arm. Halstead says that in his cases the movements of the arm were extensive and good; but, speaking from considerable experience, I may say that the movements of the arm are by no means always satisfactory. I have done this operation myself on certain selected cases, and good movement of the arm has ensued; but in my last two cases of the kind the movements were very bad, the limb being tied down by an extensive neuralgic scar, and the condition of the patient was very unsatisfactory. Mr. Treves, at the debate at the Royal Medical and Chirurgical Society, also spoke most strongly of the bad local conditions he had seen after this extensive operation. One of the main reasons you get the extensive contraction is in consequence of the large area of skin removed. Whenever a large area of skin is removed about the axilla, you are very liable to get an extensive, contracting, painful local neuralgic scar.

Finally, with regard to this very extensive operation, I may mention the remarks of Mr. Bennett May in his recent Ingleby lecture. He spoke very much as I do with regard to this operation, and he doubts very materially as to whether the ultimate results of the operation as to freedom from recurrence, are at all equal to the risks and severity of its performance.

Of course the grave question which arises with regard to this extensive operation on the breast is as to the probable prolongation of life after it, and

if a very considerable proportion of patients, as has certainly appeared in the cases hitherto reported, die of visceral cancer, one does not see that they will gain very much from this extensive proceeding. I hold no brief for imperfect operating. A great deal can be done in taking away cancer of the breast, as I shall show you, I hope, presently, without such an extensive operation as that of Halstead.

Perhaps I may now mention shortly my own general method of operating. I may repeat here what I said the other night in London. Surgeons commit an error in thinking that any one operation will do for cases of infinite variability. Is there, for instance, any kind of cancer of the breast where you might do a partial operation, removing only the disease? It sometimes happens that cancerous tumours do not grow in the breast, but in its neighbourhood, forming in accessory *mammæ*. If you can make sure that the accessory mamma is not really a prolongation of the mammary gland, I can conceive it justifiable to remove the growth only by a partial operation, leaving the breast intact. And that such practice is not altogether to be condemned, I would mention a case in which Mr. Pearce Gould performed such an operation. Two years afterwards the disease returned in the axillary glands, not in the adjacent mamma. Those glands were thereupon removed, and the patient was again free from disease; the breast remained untouched the whole time.

Let us consider another variety, namely, a case commonly enough brought before us, where an early cancer occurs in the breast with retracted nipple, and the axillary glands are not obviously affected. The operation I would advise in such a case would be free removal of the breast by an oval incision including the retracted nipple, clearing out from the axilla all the glands and lymphoid tissue, and dissecting the fascia and connective tissue cleanly off the pectoralis major. Such an operation would, I believe, completely remove the cancer without anything more extensive being necessary.

Next we will discuss a too common variety, where in addition to the cancer in the breast the axillary glands are extensively infected. This is a condition which is very often brought before us in practice, and in addition the growth may be fixed to the pectoral muscle beneath. In such cases I

advocate removal not of the whole pectoralis major, but the sternal part of it. Removal of the sternal pectoral is readily done by pushing strong scissors beneath its respective attachments, and severing by successive strokes. This proceeding adds only a little to the severity of the operation. The infected glands can be very certainly removed after such extensive exposure. The clavicular fibres of the pectoral are left in their normal situations, and can so aid in the after-movements of the arm. This is the most advanced operation I would advise in the last class of case I have described; it is very thorough, but can be quickly performed. I think I have said enough to illustrate my remark, that no one operation can be applicable to every kind of case of cancer, and the surgeon will do best for his patients if he selects in each instance that particular operation which is suited to the condition of the growth and the patient before him.

I would now say a few words regarding the question of removal of the axillary glands. I have already stated that very great differences of opinion were expressed in this matter by surgeons of eminence at the recent debate. Some never remove them at all unless they can plainly feel them enlarged and infected. This I believe is a fallacy, and I am confident, having made many microscopical and other observations, that it is quite impossible to be sure whether the axillary glands are infected or not unless the axilla is open and they are felt; and even after that you cannot always be certain, because often the evidence is merely microscopical. I have examined sufficient of these cases to feel sure that the wisest and best course for the surgeon is to open the axilla and clear out the glands in every case. If they be found to be extensively infected, remove the sternal part of the pectoralis major in order to well get at the disease. The next point we go on to consider is the operative treatment of recurrent cancer. But before speaking of this, a word or two must be said about the nature of so-called recurrence. Recurrence is a term which is used often in this connection, but it is often a misnomer. If operation is performed for cancer of the breast, and fresh growth occurs in the neighbourhood of the scar or glands in a few months, it does not mean recurrence; it means incomplete removal in the first instance, it shows that some of the infected

tissue has been left behind. Secondly, what are we to say about those extraordinary cases, which I confess I hardly knew existed in practice until I began to inquire carefully into the matter, of further growths of cancer appearing in the scar and glands some fifteen to twenty years after operation? Sir T. Smith in the recent debate, mentioned an instance where cancer had appeared in an axillary gland actually twenty-four years after the primary operation! Again, what are we to say of cases—such an one happened to me the other day—of a patient having the axillary glands and breast removed, who remained absolutely well four years, and then developed rapidly-forming ascites, cancer being found in the peritoneum? Are we to think of these as recurrences of cancer, or further outbreaks of the disease in predisposed persons? This matter has lately given rise to a great deal of debate, and much difference of opinion was manifested. Some surgeons, among whom may especially be mentioned Mr. Hutchinson and Mr. Butlin, held that such manifestations were not recurrences, but merely further outbreaks of the disease in predisposed persons. But there are these powerful, and, I venture to think, practically unanswerable arguments against these views. If these are further outbreaks of disease, how is it that nearly two-thirds of them occur in the scar or in the neighbouring glands of the area previously involved? Primary cancer of a lymphatic gland is a pathological rarity. How is it that cancer recurs so often in the bones, as the femur or spine, after removal of the breast? True primary spheroidal carcinoma in the bones, such as the femur or spine, is practically unknown. If we search literature we can find a certain number of cases of cancer of the brain or dura mater after removal of the breast; here the dissemination from the breast focus seems undoubted. A large number of cases of cancer after removal of the mamma come back to us with a growth in the lung, pleura, or liver, structures in direct lymphatic contiguity with the area from which the disease has been removed. I think these are very strong arguments indeed, and are evidence of infection having occurred from the primary focus in the breast; the cells have for some reason long lain dormant in the tissues. This theory is of the greater importance, because it urges upon us the advisability of early operation. The reflection also occurs to our minds that there

must be in these individuals a curious something or other which is inimical to the growth of cancer, and which keeps the disease quiescent in their tissues, or that the disease itself varies extremely in rate of growth, or in virulency. All such observations tend to encourage the hope that the seropathy of cancer may not entirely be a pleasing dream!

Now, what operations are to be done in recurrent cancer? It very commonly occurs that in the scar of the mammary tumour after removal you find one or more button-shaped nodules, and the very best results occur from dissecting them out by oval incisions. Some of such patients have great prolongation of life. The late Mr. Durham had a patient operated upon by himself and other surgeons some twelve times for these small recurrences in the neighbourhood of the scar; she remains well twenty-two years after the first operation. This is an extreme and exceptional case, but there are many others less striking which exemplify the good of this practice. Such local recurrences are easy enough to deal with; but when you get return in the axilla, when the arm is enormously swollen and brawny, œdematous and blue, the nerves and vein being pressed upon, what operation, if any, are we to do then? In a very large number of such cases there is no doubt that nothing operative can be accomplished. The mediastinal glands and cervical lymphatics are too often implicated, and likely enough the pleura or the liver. But there are operations advised, one of which is amputation of the arm at the shoulder. This may be justifiable for a case of extreme pain; but unfortunately, as a general rule, when the humeral joint is cut through we find cancer on the face of the stump, and this is why amputation is generally such a poor resort under these circumstances. Again, operation has been suggested on Berger's method, in which the whole limb and the scapula and part of the clavicle are removed. Two large flaps are reflected, the clavicle sawn through, the subclavian vessels tied, and the whole mass removed. My colleague, Mr. Dent, has lately done a successful operation of this kind at St. George's Hospital, in a case of recurrent cancer implicating the axillary nerves. The objection to Berger's operation is the terrible disfigurement, and there is also a more vital objection, and this is that the cancer

is usually growing in such parts as the mediastinal glands or internal viscera at the very time this proceeding is being adopted. I have taken out the sternum of four cancer patients who died with great œdema of the arm. In all of them were deposits of disease at the back of the sternum. Such extensive operations are consequently very seldom indeed justifiable. I do not think they ought ever to be performed, unless in some extraordinary and extreme circumstances. I now draw your attention to two lesser operations which may be done in these cases for the palliation of pain, which are very useful. One is the stretching of the axillary nerves, and the other is the dissecting away of cancer from them. Four years ago I treated the case of a lady with frightful pain from recurrent cancer in the axilla, and I found the axillary nerves impacted in the growth. I split the masses of cancer, and separated them from the nerves with a director. Almost for the remainder of her life the pain was strikingly relieved. This idea is not my own, I borrowed it from a case of Mr. Mitchell Banks which he published, one in which he performed much this kind of operation with great success.

Having said as much as time will allow about operations for primary and recurrent cancer, we go on to a matter which will interest you all here, more than the actual performance of the operations, and that is the prognosis in respect to these cases. One of the first questions the patient or relatives will ask you is, What is going to be the result of this? Will the operation cure the disease? Some of you may know that two or three years ago I wrote a letter in the 'Lancet' calling attention to the fact that the term "cure" of cancer of the breast by operation was not a very happy one. I believe that this my idea is also supported by the majority of London surgeons. Cancer of the breast is not like an epithelioma on an old sore of the leg or a chronic epithelial growth on the penis, which generally never return when early and thoroughly operated upon. We know what a common-sense Englishman means by the term "cure." If we remove a fatty tumour from the arm we say to the patient the disease is cured. We know, and our patients know, what this means. The disease is removed, eradicated; it will never return; the organism is left in a healthy state. I think we cannot

say that of cancer. It is true that a large number of patients after extensive operation for cancer are alive, and keep well for three years without any return of the disease. But can we say that such a case is cured? You may, indeed, tell your patient that it is an axiom in surgery that if the disease does not recur in three years it may be considered cured; but if such a patient has a recurrence in three years and six months in the spine or liver, what will she and her friends think of our promise, and what will they think of our profession as to accuracy of prognosis? Cancer is a disease which is too ill-understood to lay down prognostic laws with certainty. To state what we are not in a position to know, will shake the faith of our patients in legitimate practice. What we can tell them is that such and such measures will lead to very long freedom from recurrence, and that if the disease returns in the scar or in the neighbourhood, by repeated small operations they may have a long lease of life. We may encourage them with reports of favourable cases of freedom for ten or fifteen years, but we should not make horoscopic statements which may be falsified by events beyond our knowledge or control.

I should like to urge upon this meeting very strongly that removal of the breast on the modern plan, with dissection out of the outlying lobules of the gland and the pectoral fascia, and perhaps the removal of the sternal part of the pectoralis major, is not an easy and simple operation; it requires a very great deal of care and skill. A large number of operations are attempted for cancer of the breast which are notoriously incomplete, the dissection is not done thoroughly as it ought to be, and infected tissue is left in the wound. The clearing of the axilla from glands is seldom done properly, because the incision is seldom made large enough. Inexperienced operators are satisfied to make a small incision, and scratch and tear with their fingers through this instead of laying the parts open to observation and thorough surgery. Another important matter is rapidity in operating. Some surgeons tell us that they take one and a half to two hours over removal of the breast! Such a lengthy operation, in my judgment, adds very seriously to the danger of the patient. The risk of bronchitis in an aged person from such a long operation on the thorax is very grave. Another point of vast importance is the question

of the anæsthetic. I am sure in operating for removal of the breast it is a very bad thing to keep a patient heavily drenched with ether for a long time; I always request the anæsthetist to give the patient gas and ether at the start, and towards the middle gradually substitute chloroform in the smallest quantity possible to keep up anæsthesia. If this is properly done, sensibility will be returning as the dressings are applied. Warmth of the room and warm clothing to the patient are also of great importance, and I need hardly say that the strictest care must be exercised in asepsis. When I speak of rapidity in operation I mean the rapidity conformable to safety and thoroughness. I have seen surgeons waste many precious minutes in talking, preparing instruments, in shifting the patient about, all of which should have been done before the operation, not when the patient is under the anæsthetic.

In concluding this sketch of the operative treatment of cancer of the breast I would urge very strongly a point which I hinted at before. Believing as I do that recurrence of cancer is due to dissemination from a local focus, the great secret in getting successful results and long periods of immunity from recurrence—and it is the factor I would insist upon more than any other—is early operating. This is the one thing we all ought to have before our minds. It so often happens, though perhaps not so often in modern days, that out of mistaken kindness cancers are covered up with plasters and concealed, and patients are encouraged to take this or that drug until the cancer becomes inoperable. In cancer of the breast, if the disease is removed freely when it is as big as a nut, we should have still better results in the treatment of this formidable malady than we are obtaining at present.

I have only given you a bare sketch of a very vast subject. If I had gone thoroughly into all the subjects which I have brought before you I might have been here for several hours. I only hope you will show your appreciation of the remarks I have made by asking me any questions upon matters which I have not made perfectly clear.

In answer to questions, Mr. Sheild said the president's criticism of the terms "complete" and "incomplete" was very proper; the same objections had been repeatedly made, and had occurred

to his mind. He only used the terms in his remarks, because they had been adopted largely; not because he thought them right. He thought the terms "lesser" and "greater" operations were preferable. Though he had spoken of Halstead's operation as being too extensive for ordinary cases, he could conceive instances where the operation would be justifiable. For example, in a robust woman with a cancer adhering to the pectorals, he would say Halstead's was a suitable operation, but if he were asked to perform the operation on a feeble person with bronchitis or cardiac disease, he would not do so. The operation should not be done as a matter of ordinary routine. He used the term "Halstead's operation," but we should remember it was founded on the observation of the pathologist Heidenhain. Regarding the subsequent movement of the arm after an operation involving the clearing out of the axilla, it was his invariable practice never to bind the arm to the side after operations on the breast. He always left the arm in a sling; and after ten days, as soon as the wound was healed, he instructed the nurse to raise it from the side very gently and insert cushions beneath it. In this way he had generally got good movement afterwards without the wearing of a splint, which he thought would be rather irksome to the patient. There would be no objection to trying the splint in cases where the axilla was greatly opened, and especially when muscle was removed. He would be glad to try anything to obviate contraction in these cases. He thought the splint mentioned by the chairman was by no means new, as he (Mr. Sheild) had in a manuscript book, a copy of a very similar splint advocated by an American surgeon to prevent contraction after burns. As to the use of Coley's fluid in inoperable cancer, the matter passed through his mind, but he did not touch upon it because he was dealing with the operative treatment of cancer of the breast; and other matters, as electricity, drugs, &c., were too numerous even to mention. However, he was in a position to give them definite information. Two years ago he published a very remarkable case of recurrent sarcoma of the breast treated with this fluid. Two large nodules of the growth disappeared entirely under its use, but it unfortunately happened the remedy also killed the patient. There was a risk of destroying the patient in order to cure him of his

disease! He could only say that it was a most dangerous treatment. He had a patient at St. George's Hospital at the present time with two large inoperable recurrent sarcomata on the back. He had removed them twice, and a senior colleague had operated on them three times. They were now so large and deep as to be no longer fit for operation, and he was treating them with Coley's fluid. He injected the larger tumour with three minims of this preparation, and the effect was nearly to kill the patient, for within an hour of the injection she had a terrible rigor, became almost pulseless, passed her motions in the bed, and was only kept alive by the free use of brandy. The tumour he injected sloughed somewhat after acute inflammation, and the other a little diminished in size; he was intending to inject it again, and the ultimate results would be probably one day published. Coley himself had laid it down that the injection of this fluid had done good in spindle-celled sarcoma; the results in cancer, even according to the inventor, had been poor. Other surgeons had come to the same conclusion. He (Mr. Sheild) had employed it in three carcinoma cases, and in all of them he had had to leave it off; no good was done. He questioned whether the fluid would ever be curative in carcinoma, but in spindle-celled sarcomata it might be of some benefit if employed with the extremes of caution. A method of treatment, on which he was surprised no question had been put to him, was the removal of the ovaries in recurrent cancer of the breast. A certain number of cases had been done, notably by Beatson, of Glasgow, and remarkable results had ensued, namely, great diminution of pain and shrinkage of the cancerous growths. But he (Mr. Sheild) thought the operation should only be performed in rather young women. He did not see how it was going to benefit a patient getting on in years, whose ovaries were inactive, and not, therefore, influencing the vascularity of the mamma. He might mention that he had information to the effect that recurrence of growth was already manifest in some of the published cases of success, and he knew of two cases of "cure" not published where the removal of the ovaries had had no effect whatever on the disease! Still he thought it was justifiable to do the operation on a young woman with inoperable breast cancer, but only after very grave deliberation and

consultation, for the treatment was open to wide abuse by design or ignorance.

As to the question relating to the microscopical characteristics of recurrent cancer, in the majority of the cases he had examined the fresh growths were invariably of the same type as that in the breast, namely, spheroidal-celled carcinoma. In cancers of duct origin the epithelium was of the columnar type, resembling that of the original disease. A question had been asked as to what was the longest period of time on record without a recurrence of cancer after operation. This was a very difficult query to answer accurately, because it depended upon the truthfulness of humanity, which in the matter of successful cases in one's own profession was sometimes a little uncertain. The longest period of freedom from recurrence which was mentioned in the recent debate was twenty-four years, but even after that time the disease returned in a neighbouring gland. He could not now remember the longest period in the cases he had collected, but there were several of from fifteen to twenty years. The longest period of freedom in his own experience was fifteen years. He knew a lady who was operated upon for cancer of the breast fifteen years ago, in whom the axillary glands were not touched, and she was still said to be in perfect health. As to whether recurrences were more likely to occur in an old or a young person, he would say, as a general rule, that the older the person the slower would the cancer grow. In an old wrinkled woman of seventy from whom cancer had been removed, a recurrence would be very long delayed and very slow growing, whereas in a young robust and plethoric woman, more especially one who was pregnant or lactating, the disease would return with virulence and rapidity, so much so that it may seem worth while operating. But there were exceptions to this, and exceptionally a very bad type of primary cancer was followed by long freedom after operation. This brought him to say that there was no doubt a great deal more depended on the curious vagaries of the rates of growth of cancer than was generally believed. Extraordinary examples had been related of spontaneous retrogression of cancer. Professor Bennett, of Dublin, kindly informed the speaker of the case of a near relative of his own; she had cancer twenty years ago, and it simply withered away. If this case

had been operated upon, he invited them to think, what a boast there would have been of the successful results of the special operation performed. Therefore they were obliged to take into account the vagaries and eccentricities of the disease, because it differed extraordinarily in different individuals. He had been asked how long an operation for removal of cancer of the breast took to perform? That of course must vary according to the complexity of the case, and especially the experience and skill of the operator; but it ought to be a case of considerable difficulty to take more than half an hour. The growth ought to be removed, and the wound commencing to be united by that time. He very much deprecated operations one hour and upwards, for they exposed the patients to considerably increased risk.

[Mr. Sheild was heartily thanked at the conclusion of the meeting.]

NOTES, ETC.

When may Women with Heart Disease Marry?—Dr. Kisch ('Therapeut. Monats.,' February, 1898) said the chief points to be considered are: (1) the kind of heart disease; (2) its duration; (3) the presence or absence of compensation; (4) the general health; (5) the social position of the patient.

(a) They may marry if the disease is not of long standing, and compensation is good, and the general health not undermined. They will have during pregnancy, and still more during and after delivery, many troubles due to their heart, but in by far the greater number of cases there will be no danger to life. This applies to well-compensated mitral regurgitation and stenosis, aortic regurgitation, fairly marked sequellæ of pericarditis, and to muscular degeneration if not too far advanced. The patients must also be in a position to spare themselves bodily exertion as much as possible during pregnancy, to avoid mental excitement, and to have constant medical supervision.

(b) The prognosis is not so good if the patients are very anæmic or nervous, or advanced in years, or if the valvular disease is congenital or acquired

in childhood. In these cases the physician should advise against marriage, or at any rate point out that the disease will almost certainly become worse after marriage.

(c) Marriage is to be absolutely forbidden as dangerous to life when compensation is failing, or when there is advanced muscular degeneration. In all cases where there is dyspnoea, palpitation and quickened pulse on slight exertion, or marked oedema not disappearing after rest in bed, when there is tendency to arrhythmia, scanty urine with albumin, and attacks of irregular small pulse, coldness of the extremities, nausea, dyspnoea, syncope, &c., marriage is dangerous, whether the cause of the symptoms be valvular disease, diseased arteries or cardiac muscle. Even those for whom marriage is allowable must follow certain rules strictly.

1. Coitus must not be frequent, and must be continued to the end of the orgasm, otherwise reflex heart troubles and depression result.

2. They must not have more than one or two children, as the strength of a diseased heart diminishes with every pregnancy in geometrical progression. If this rule is followed induction of premature labour will be luckily seldom necessary, since when it is the results are very unfavourable.

St. Louis Medical and Surgical Journal.

GRAHAM, discussing at length the prognosis of heart disease, considers that in early life valvular lesions are the result of endocarditis, and the prognosis during the attack depends largely upon the nature of the infective agent. If due to rheumatism the immediate prognosis is favourable, but there is a liability to its recurrence. When as a result of scarlatina it is not likely to recur, and hence the prognosis is more favourable than when due to rheumatism. Murmurs occurring during an attack of rheumatism frequently disappear, so the attending physician should not alarm the friends by predicting serious heart disease. When, however, the murmur lasts from the beginning to the end of the attack, it will never disappear in after life. The question often arises, How long will complete compensation last after it has once been completely established? To determine this it is necessary to find out if possible the extent of the lesion, the length of time during which it has existed, the changes which have taken place in the heart itself

as a result of the lesion, the condition of the other organs of the body, the temperament, mode of life, habits and calling of the patient, hereditary tendencies and presence or absence of other disease. After a full discussion of the physical signs of the various lesions, the comparative gravity is stated in general terms to be, commencing with the gravest, tricuspid insufficiency, aortic insufficiency, mitral stenosis, aortal stenosis, and mitral insufficiency. The social condition of the patient is an important factor in prognosis. If he is of regular habits, does not use tobacco, is either a total abstainer or a very moderate user of alcohol, possesses an even temper, is one whose calling requires a moderate amount of regular exercise, who lives among healthy surroundings, and who has freedom from worry—he stands the best chance of a long life. Valvular lesions which originate after the middle period of life are usually the result of various forms of degeneration, and are often the direct result of an infection. When systolic or diastolic aortic murmurs occur in an elderly individual, and are accompanied by fever, a very guarded prognosis should be given as to the immediate results. There is a class of cases in which there is serious disease of the myocardium, when no valvular murmurs can be heard, and may be due to local or general fatty degeneration. The diagnosis of such conditions is extremely difficult, and on that account a prognosis is not usually made. One of the most frequent questions asked by a patient and his friends is with regard to the possibility of sudden death. If the patient can be assured that in his particular form of disease sudden death is not probable, it will have a wonderful influence in relieving his mind. In all cases of advanced disease there is a possibility of sudden death, but it may be safely stated that there is no danger from sudden death, in the sense of falling down dead, in any of the valvular lesions except aortic incompetence. In all the class of cardiac neuroses, palpitation, brachycardia, tachycardia, the most careful examination should be made to exclude organic cardiac disease before a prognosis is given. It will then depend upon whether the cause can be removed or not. Arrhythmia is more grave than palpitation.

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A CLINICAL LECTURE

ON

PENETRATING WOUNDS OF THE CENTRAL NERVOUS SYSTEM.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, London, Dec. 17th, 1897,

By VICTOR HORSLEY, M.B., B.S., F.R.C.S., F.R.S., &c.,

Surgeon to the Hospital, and to University College Hospital.

GENTLEMEN,—The surgical subject that I wish to lay before you to-day is a discussion of the wounds and lacerations which penetrate and otherwise damage the central nervous system. We have before, in these lectures, discussed the question of new growths, with the question of compression-myelitis in the spinal cord, and other compression effects in the cerebrum and cerebellum, but we have never considered the large class of conditions under which either a recent wound or laceration of the central nervous system may be caused, or in which we have to treat the effects of a healed wound or laceration.

Let us first review the ways in which wounds or lacerations of the central nervous system are brought about. They are usually produced by some sort of missile, either a bullet (gunshot wound), or stones and other objects falling from a considerable height, these latter, of course, often moving with a great momentum. Rarer cases are those in which the brain is wounded through a thin portion of the skull, such as the temple, by a sharp stabbing instrument, like a dagger, and then we have those relatively not infrequent cases in civil practice in which a child holding a pencil or pipe falls down upon it; or where a person with a considerable amount of temper thrusting an umbrella or stick at another person drives it through the orbital plate and the frontal bone, and so causes a penetrating wound of the brain. We

must also bring into the same list simple contusions and lacerations of the brain.

Now, in the first place, we have to remember that under no circumstances can we ever have a penetrating wound of the central nervous system without at the same time damage being done to certain soft parts. That of course brings about many important complications, notably in the instances of gunshot wounds. I show you in the lantern the photograph of a recent gunshot wound made with a very small calibre (.22) bullet. The skin has been removed, and you see the amount of bruising and the punctiform hæmorrhages all round the actual bullet channel. The local disturbance is greater in the aperture of exit, the photograph exhibiting the amount of laceration in the muscle. I now show you a deeper dissection of the first view, after clearing away the damaged muscle, and showing the wound in the bone. The wound in the bone is marked by a fringe of fragments of bone which are driven in. So much for gunshot wounds and damage done to the superficial parts.

Now we come to the consideration of falling bodies. Here we have a photograph of a very striking example of a wound produced by a small body falling from a great height—a piece of brick falling from a height of eighty feet—and consequently acting like a large bullet, penetrating the skull. This portion of brick struck the cranium just opposite the very top of the fissure of Rolando. There are many interesting points about the case which I cannot go into now, but I must speak of the secondary septic inflammation set up in this instance, because it demonstrates in a striking manner the chief among the immediate consequences of a penetrating wound, viz. hæmorrhage. I would draw your attention to the fact shown in this photograph that the membranes have been stripped off from the two central gyri with the utmost ease, but that the attempt to peel them off from the rest of the brain has torn the surface owing to adhesion. The patient was treated many years ago at University College Hospital, and died of septic meningitis. But, as you have just seen, the membranes covering the central gyri have not taken part in the meningitis. The reason that they have not done so is that you have, as in all penetrating wounds of the brain, hæmorrhage descending in the corona radiata, and therefore in

conjunction with the hæmorrhage in the pia mater, completely cutting off the circulation of the part of the cortex beneath which the dissection has occurred. In the two central gyri, the vessels were thus completely thrombosed, and could not take part in the general inflammatory process.

This second photograph is a good instance of contusion or laceration of the cerebrum, such contusion being conditioned partly by a local fracture and partly by the shape of the brain within the skull. The cortex in some places is much torn.

The next question we have to think of in connection with these penetrating lesions is the one I referred to in speaking of the character of the aperture in the skull—namely, the associated destruction brought about by concomitant complications of the injury. Take a bullet, for instance. When we are dealing with a bullet that has passed through the soft parts of the skull, we have in our treatment to pay as much attention to the foreign substance brought in with the bullet as to the bullet itself. I show you one of Mr. Boys' photographs of a bullet of small calibre and high velocity after it had passed through a glass plate; you will notice the cloud of *débris* surrounding it, and moving at the same rate. Exactly the same thing happens in regard to fracture of the skull. In fracture caused by a bullet you often find in the brain a quantity of *débris* extending practically the length of the track of the bullet. That is an important practical point. These accessory foreign bodies have imparted to them a velocity which bears a very considerable proportion to the velocity of the bullet or projectile itself; and that is the reason the *débris* gets carried almost as far into the brain as the bullet itself. I have proved the same thing in other ways by firing a bullet through a plate of bone (the body of the scapula) behind which was a large mass of modelling clay, and on examining the clay afterwards I found in these experiments that fragments of bone were imbedded in the clay right up to within half an inch of where the bullet was lodged, showing that the fragments of bone had had imparted to them practically the same velocity as the bullet. Again, when we are dealing with bullet wounds of the head, we have to remember that the greasy hair, which is a very septic material, is carried in the same way a long way into the hemisphere.

We now come to the consideration of the effect

of a penetration wound of the brain. These effects are to be conveniently divided into two heads:—(1) immediate or (2) remote. If we have a patient who has been stabbed in the temple with a dagger, he suffers no immediate effects beyond the fact that the force of the blow has stunned him. If, on the contrary, we have a person who has been shot through the head with a rifle bullet, or struck violently on the head with a cricket ball, we have the patient apparently killed on the spot. Therefore we must discuss first the immediate effect of the wound of the brain in producing death, so that the first question we may have to answer in such a case is, "To what is that death due?" Now, for a long time death from all forms of acute concussion of the brain was attributed to failure of the heart. It was supposed that somehow the heart was affected, and that the patient died of syncope there and then. We have now learnt that the danger to life is not through that at all, but through the respiratory centre. I show you here a few of the photographs of the tracings obtained by Dr. Kramer and myself upon which this fact is based. As a preliminary question we will take the difference between the kinds of projectile that pass through the brain as concerning, firstly, the effects produced by their velocity, and secondly, the presence of the foreign substances in the brain. Do we have produced in the brain a degree of destruction proportionate to the velocity of the shot? In order to arrive at a determination of this it is necessary to devise some apparatus which will register the effect of a bullet which has passed clean through a substance, and also the effect of a bullet which, starting with the same velocity, nevertheless does not get through the substance fired at. If the bullet passes clean through the substance and goes out on the other side, it is clear that only a small portion of its energy is used up in the target; in other words, only a small portion of its energy is converted into shock or is actively destructive within the brain. If, on the other hand, a bullet of the same weight and the same initial velocity passes into the target and stays there, all its energy must be used in producing shock. This may be shown experimentally by an instrument I devised, by which it is found that when a small bullet passes clean through the skull there is only one third the shock produced by a similar bullet,

which lodges in the skull on account of it having expanded. It is conceivably possible for a small-bore bullet with a high velocity to pass through portions of the central nervous system without producing much shock; but when these bullets pass through the skull and brain and everything else, they cause sudden death under certain circumstances. What are these circumstances? In the first place, the fact of their causing death is due, as I stated just now, to failure of the respiratory centre. How that failure is brought about by the hydrodynamic force which is developed within the wet tissues was shown by Huguier and Kocher. If you take two skulls of equal strength, fill one with water, closing the foramina with wax; and leave the other empty, and then fire bullets of the same velocity at both, you will find that the bullet simply penetrates and passes through the empty skull, whereas in one filled with water there will be separation of the bones and bursting of the skull. That is simply due to the fact that to the particles of water there is communicated the velocity of the bullet in the skull, and therefore they become moving particles with great hydrodynamic power, splitting open the sutures in the manner that you see. The brain is incompressible, and so, of course, are the fluids which fill its cavities, hence, with the brain *in situ*, the effect on the skull is the same as if it were filled with water; we have the same lines of force spreading through the whole brain. Before you, you have an example of the effect of that in the shape of a brain of a dog, through the fronto-crucial region of which a .22 calibre bullet has been fired. In the cerebellum and medulla are numerous petechial hæmorrhages, as well as bruising of the occipital lobes, and of the membranes in the sides of the fourth ventricle. In this portion of the medulla is the respiratory centre. Death is consequently due not to syncope, as used to be thought, but to paralysis of the respiratory centre, which is proved to you by the accompanying tracings taken from animals shot in that manner. Here is such a tracing; upon it is also shown the electric signal mark, showing when the bullet left the muzzle of the pistol, which is practically the moment when it entered the brain. You observe that the respiration stopped instantly, while the heart continued beating almost normally. The rest of the tracing shows the record of the movements of artificial respiration employed to restore

the animal. Here is another blood-pressure tracing showing very little diminution of pressure; in fact, the heart-beat at the moment is practically unaltered, and yet, in the absence of artificial respiration, the animal died. Here, then, 'we have the first demonstration that immediate death in cases of penetrating wounds of the brain from a missile are due to failure of respiration. This is another tracing where the respiratory centre was not so seriously affected as to cause immediate death, but became extremely irregular in action. One more tracing bearing on this point will not be out of place to illustrate the question of treatment of sudden wounds and of shocks to the brain. If the view I have enunciated is correct, the deduction that artificial respiration ought to bring back normal respiration movements, provided efficiency of the respiratory centre is not permanently destroyed by the shock, must be true. You see here that the blood-pressure curve was falling when the bullet entered the brain, and artificial respiration was commenced. For some time afterwards there is seen to be no attempt at natural respiration, but the blood-pressure gradually rises to 165 mm., which is rather above the normal. As the artificial respiration goes on, slight automatic movements of the chest are seen, and ultimately the artificial respiration is left off, and the animal is then found to be beginning to breathe again.

There are other immediate effects also which are of great practical importance. The first is that we cannot have any penetration of any portion of the brain in any direction without the general destruction of two things as far as the nervous system is concerned—destruction of centres, and destruction of fibres. Destruction of fibres is the most important question practically, and I can best illustrate that by one of a series of cases which have recently come under my notice, in which a gentleman shot himself, the bullet entering just above the edge of the hair-line. The bullet was a heavy one—about .38 calibre and of considerable size, and the major portion of it took a course, as shown by the aluminium probe, straight back to the occipital region. But when it entered the skull it struck the bone at an angle, and broke up on the edge of the very hole it had made. Consequently in the lacerated brain beneath the aperture of penetration were a number of frag-

ments of bullet, and one very large fragment had taken the course indicated, penetrating the cortex and glancing downwards to the base of the skull. Now, that patient was rendered immediately and completely hemiplegic. Why was he hemiplegic? To answer this question it is only necessary to map out the course of the bullet. It penetrated the front of the centre for turning the head and eyes, then it travelled through the very middle of the corona radiata centre of the parietal lobe. Following this direction of the bullet on this cast of the hemisphere we see it travelled through the arm-centre fibres. You may ask how then did it affect the leg centre and the face centre? Here we have the operation of the other factor, of which I have already spoken in relation to a falling body penetrating the skull, namely the consequent dissecting hæmorrhage. Hæmorrhage occurring in the coronal radiata centre always dissects its way among the fibres. Thus in this case there was actual destruction of a few fibres from the passage of the bullet, but this was followed by an extravasation of blood pushing aside the fibres and tearing them through. After removing the *débris* and washing out the injured brain-tissue with strong perchloride lotion and passing a horsehair drain through the track, the patient, nevertheless, ultimately made an excellent recovery. He has practically recovered power in the leg and completely in the face, showing that the hæmorrhage had been pressing upon the fibres (supplying these parts) more than actually tearing them across.

The first thing, then, that we have to do in order to estimate the immediate effect of damage done in a penetrating injury is to localise the passage of the instrument or missile causing it as far as its relation to well-known centres and fibres is concerned. That is all very well when we have a simple case like the one I have described—where we have such definite paralysis which was easily mapped out and accounted for, but the task is very difficult when the course of a wound in the frontal region (which is very often the seat of entry) is directed downwards. Even when the position of a foreign body, *e.g.* a bullet, has been shown by the Röntgen rays, it is not possible very often to reach it, because we so constantly find the line somewhere about the sella turcica and the optic chiasm. I need not dwell any more upon this point of

actual destruction of centres or of fibres, because the detailed diagnosis of the same must always depend on a knowledge of the precise position of these centres and the course of the fibres passing from them through the substance of the hemisphere.

We now must enter upon the consideration of the occurrence and treatment of hæmorrhage at the time of the accident. Here we enter upon what is often a difficult subject, surgically speaking. If we are dealing with a penetrating wound of the temporal region, as for instance from a knife stab, then the patient may, within a short time of the accident, become unconscious and die, unless trephining is resorted to very early. Now, the patient may, of course, from accident have suffered from a wound of the meningeal artery, but a wound of this artery, to which so much attention is given in surgical works, is not a serious accident, or not serious in the same sense that a wound of the middle cerebral (which may equally be the source of trouble) is. The reason of this is that the effects of a wound of the middle meningeal require time to develop. The blood is extravasated between the dura and the skull, and the resistance it has to overcome in stripping the dura from the skull, and the normal pressure within the dura, make such extravasation a slow process. But when one of the main cerebral arteries is wounded, then the blood is rapidly poured out and quickly diffuses itself in every portion of the sub-dural space. Then we get very rapid compression, and if it is not speedily relieved the patient dies; so that practically, as regards hæmorrhage, the first point to decide is whether it is likely that one of the cerebral arteries is wounded. If it is (and I will take up the question of treatment as we go along) it may be difficult to find where it is wounded, but it is urgently necessary to look for it. The artery must be wounded at or in the line of the point of entry, and therefore a simple enlargement of the opening will meet the case. But the hæmorrhage, as a rule, is rarely due to wounding of the main cerebral arteries. Hæmorrhage, of course, invariably complicates all penetrating and lacerating wounds of the brain, but it occurs under two circumstances beyond those I have already described. In the first place it is usually due to the wounding of a large number of small arteries which commonly does not call for surgical interven-

tion. But hæmorrhage of a different kind may result from penetrating wounds, which has nothing to do with the arterial stream at all. I refer to injuries of either the lateral or other sinus, or veins leading to it. For instance, the large vein which leaves the lower part of the temporo-sphenoidal lobe and enters the lateral sinus is not infrequently wounded, and the hæmorrhage from it is very severe. When the skull is opened the blood may come out in a fountain, just as if the sinus itself was wounded. The treatment of a wounded cerebral artery is simply to tie it on the cardiac side of the wound by dipping with a large sharp aneurysm needle under the vessel a fine horsehair ligature, but it is less easy to localise the often violent hæmorrhage which comes from a wound of this sort. To deal with it adequately, the first point to be noticed is the colour of the blood. If it is dark, and therefore venous, then you know from the vigor of the outflow that it must be from the sinus or close to it. Therefore the first thing to do is to press aside the brain and push your finger along the course of the sinus until you arrive at the opening; then with the other hand you can clear out the clot by washing, after which, on removing the finger cautiously, a spurt of blood will reveal the wound. A wound in the sinus is readily sutured, and in many cases a perfectly aseptic plug will settle the whole difficulty.

It is necessary to now consider inflammation as a possible complication of penetrating wounds of the brain. The specimen I showed you at the beginning of the lecture was an ordinary traumatism resulting in septic meningitis from neglect of antiseptic precautions at the time of the injury. Nowadays, of course, such a *contretemps* is fortunately rare; but it may be thought inadvisable to push disinfection in some cases. Personally, however, I should not like to accept the responsibility of any case unless I was sure that I had washed all the lacerated brain substance thoroughly with perchloride of mercury lotion of 1 in 1000 strength. In the case of all extensive wounds it is best to open up the region widely, as it is difficult to recognise the extent of the mischief. If this is not secured, then there may follow, not acute septic meningitis, but a very slow chronic form of abscess of the brain. A striking instance of this occurred to me recently in which the patient was a boy who was chaffing his brother while the latter was hold-

ing a toy pistol in his hand, of a .22 calibre. By way of reply he pointed the pistol at his brother and pulled the trigger, with the result that he shot his brother through the thin squamous portion of the temporal bone. The patient was seen immediately after, when he was perfectly conscious. The wound was promptly trephined, the fragments of bone and hair were removed, and it was, I am informed, carefully disinfected, but the bullet was not found, though the external wound healed up by first intention. This happened on Christmas Day, 1896. After the wound healed the boy got up and went about, and for months the only thing noticed was that he was not quite as bright as usual. He then got rather apathetic, and began to lose flesh. His case was reported to me by letter from his medical attendant who told me in June, 1897, that the boy was beginning to develop nystagmus, and that in July he had got much worse. He was then sent up to London to be under my care. When he arrived, I found that there was very slight right hemiplegia, or rather hemiparesis, with a certain degree of loss of tactile sensation, double optic neuritis, and above all there was what I believe is an unfailing symptom of chronic cerebral abscess, a subnormal temperature. On examining the wound, I found that the scar was bulging and very tense, and therefore formed the opinion that a chronic abscess had formed in connection with the unfound bullet. Therefore, after thirty-six hours preliminary disinfection of the scalp, I opened the skull at the point of entry, and found an extremely tough cicatrix that went through the cortex to a depth of an inch, and situated just above the sylvian fissure. On following it down, I found there was a sac of an abscess at the end of this cord of cicatrix. This abscess being so chronic, I ventured to treat it in the following way, namely, by letting out the pus, then taking the pyogenic membrane between two pairs of forceps, and then steadily pulling with considerable force (about thirty pounds). By these means the abscess sac drew out exactly like an hydatid membrane. Then I found the bullet was lying on the further side of the sac, between the sac and some lacerated brain. There is no doubt that many cases of penetrating wound of the brain, especially those which have been caused by an umbrella or stick forced through the roof of the orbit, terminate similarly with chronic

cerebral abscess; and there can equally be no doubt I think that the lesson to be derived from such cases is that we must prosecute our search whenever a foreign body enters the brain by cleansing as far as possible its track after we have removed the pieces of bone and other *débris* around the orifice of entry. Though, of course, we are naturally anxious to do as little further damage as possible, it is an urgent necessity to follow up as far as possible the foreign substances, and the question arises, what means have we at our disposal to avoid adding to the injury? I think the simplest is Flührer's aluminium probe, because you can let that fall gently along the track of the bullet without the danger of rupturing the delicate structures which attends the use of a heavier probe. Or we can use the electrical probe so as to recognise in the telephone when the bullet is touched.

I have so far referred to the immediate effects of lacerating wounds of the brain, and now come to the remoter effects. The most disabling of these are sensory paralysis and hemiplegia. A very practical question is the possibility of helping the patient as regards his hemiplegia? We have seen what the hemiplegia is chiefly due to, namely, rupture of the fibres by hæmorrhage. It is quite clear that fibres which are torn across cannot be reunited; it is also clear that blood extravasated in the walls of the wound cannot be removed. Therefore its absorption is all that can be expected.

Recovery of power from absorption of the clot under these circumstances occurs early. Within about three weeks of the accident the patient already begins to recover power. Then, of course, the further question is asked whether anything can be done in the way of helping the damaged cortex to recover its functions. There is no way except by stimulating the centres into action, and it is very doubtful how far it is justifiable to attempt to excite the brain. This is a very serious matter, because in many of these instances we know there is a foreign body in or near the cortex, and therefore the patient is liable to traumatic epilepsy. If a faradic current is employed to excite the cortex in the neighbourhood of the wound to greater activity there is considerable risk of aggravating the traumatic epilepsy, or the condition which causes it. So that it is far better

to leave things entirely alone, at any rate for four to six months after the injury.

I have spoken chiefly about wounds of the cerebrum, but what I have said applies equally to the wounds and injuries of the cerebellum.

We now come to the spinal cord. The cord is often wounded by bullets, and also has not rarely been wounded by a stab from a knife when the knife blade has passed between the laminæ of the vertebræ. The relative possibility of this occurring is well shown by a dissection shown in one of Professor Fraser's photographs, exhibiting the spinal cord *in situ*. The cord, especially in the cervical region, fills up a great deal of the space of the neural canal, consequently a penetrating body of any breadth above a centimetre is certain to seriously damage a portion of the cord. This case now in the hospital is an example how these injuries commonly occur. The case is that of a young fellow who in San Francisco was successful in obtaining an appointment, whereupon a disappointed competitor shot him in the back. The bullet penetrated the laminæ of the second lumbar vertebra, and was shown by the Röntgen rays to be embedded in the body of the vertebra. In reaching the spot it passed through the right half of the spinal cord. In the great Army Museum at Washington is a parallel case of a person who was summarily shot, showing a bullet wound of the cord; the bullet passed through the cord and inflicted a simple restricted division of a certain number of fibres. It is clear that in the neural canal we have not the same conditions as in the brain—we have not the same large mass of material; in other words, the bullet wound of the cord produces a very localised lesion, but one which affects the nerve-supply of a large area of the body. I wished to show you this case to demonstrate the localised effect of such an injury to the cord. He has corresponding motor paralysis, and I may say, just to conclude the case, that he was operated upon in San Francisco, the position of the bullet having been successfully localised by the Röntgen rays, and the bullet was removed. The question is how far these cases can improve. I think they in no wise differ from the injury which is effected on the cord by the sharp edge of a laminated vertebra in the case of a fracture, such as I show you here. Where you see the cord tissue has been divided and torn across and lacerated, there is in all these

cases a certain amount of hæmorrhage up and down the substance, which will be absorbed, and so far slight improvement will set in, but where the fibres of the cord are damaged there seems to be no chance of their subsequent union. When the question is decided as to how far the fibres of the spinal cord can be got to unite, in the same way as the fibres of a peripheral nerve apparently do, we can speak definitely of the prognosis in these cases, but undoubtedly the only duty which remains to the surgeon under such circumstances is to make sure that there is sufficient exit for extravasated blood from the dural sheath. As to the subject of treatment of the cord by attempting to arouse greater activity in it by electricity and otherwise, that is extremely justifiable in the case of the cord, whereas for other reasons which I have mentioned, quite different reasons, it is not justifiable in the case of penetrating wounds of the brain.

Therapeutics of Carbuncles.—Dr. S. Rosenbaum, in the *New York Medical Journal* of June 11th, in an article on carbuncles, gives the following method of treatment as pursued by himself:

Fold a piece of aseptic gauze until it forms a thickness of six or eight layers, the surface area to be somewhat larger than the carbuncle to be covered. The gauze is first thoroughly saturated with Thiersch's solution, then covered with a layer of 10 per cent. ointment of ichthyol, and then applied to the carbuncle. A piece of rubber protective, large enough to overlap the gauze, is now placed on the same to keep in the moisture. A layer of cotton wool is placed on the protective, and then the bandage is applied and allowed to stay two days. When the patient returns to have the bandages changed the cores are found to have separated from their respective walls, and at the next re-dressing, which is in a few days, they are found entirely separated, and can be easily and painlessly removed. At the next visit granulation has passed the primary stage, and healing quickly results, leaving an almost invisible scar. The only constitutional treatment necessary is the administration of cathartics.

The writer claims for this method the following advantages:—(1) Painlessness; (2) quickness of healing, more so than with other methods; (3) no scar or cicatrix.—*Medical Age*, July 11th, 1898.

A POST-GRADUATE LECTURE ON ANTE-PARTUM HÆMORRHAGE.

Delivered at Charing Cross Hospital, June 30th, 1898,

BY

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LADIES AND GENTLEMEN,—I have chosen for our consideration to-day the subject of Ante-partum Hæmorrhage, and more particularly the hæmorrhage which occurs in the later months of pregnancy. Just, however, to clear the ground, I would like to mention those cases of hæmorrhage in the early months of pregnancy, some of which may continue into the later months.

The causes of hæmorrhage in the early months of pregnancy may be divided into causes *due to the pregnancy itself*, and causes *complicating the pregnancy*.

Those *due to the pregnancy* may be such as threatened abortion, which may be inevitable; incomplete abortion where something is left behind; and missed abortion where the ovum is dead, but has not come away. Then, any form of molar pregnancy, either an apoplectic ovum or the more organised and somewhat larger fleshy mole, or the hydatidiform degeneration of the chorion. There is a further cause of hæmorrhage, which I think is commoner than the other—decidual endometritis, affecting more particularly the lower zone of the uterine cavity. Any of these, and perhaps deciduoma malignum should be added, may produce hæmorrhage in the early months of pregnancy, and are directly due to the pregnancy itself. Then there are certain *causes of hæmorrhage* which may *complicate pregnancy*. The commonest of all is mucous polypus of the cervix, and then hypertrophic erosion or adenoma of the cervix, which usually only causes hæmorrhage on coitus; malignant disease of the cervix and the vagina, and certain ulcerations, specific and non-specific. Vascular caruncle of the urethra is generally given as

a cause of hæmorrhage, but that again practically only causes hæmorrhage on coitus.

In the later months of pregnancy any of these causes which I have mentioned, as complicating the pregnancy, may be present. The causes due to the pregnancy itself may be divided into three. First the so-called "accidental hæmorrhage," then the "unavoidable hæmorrhage" of placenta prævia, and thirdly deciduoma malignum. Deciduoma malignum is probably a sarcoma which is affecting the uterus, and which has secondarily affected the decidua and perhaps the placenta. German writers seem to think it is a primary disease of the decidua and placenta, but it is indistinguishable in the great majority of instances from an ordinary sarcoma. The probability is that it is very rarely a true primary deciduoma.

This afternoon I want to speak specially of *accidental hæmorrhage* and *unavoidable hæmorrhage*.

Accidental Hæmorrhage—probably a good deal commoner than the other variety—merely means hæmorrhage from a partially separated placenta which is attached in a normal position, that is to say *not* at the lower zone of the uterus. We do not know the cause why some placenta should become detached and others not, but we know certain things excite hæmorrhage of this variety, such as blows or falls, or mental disturbances such as sudden shock, or fright, or emotion. But there are also definite predisposing causes, such as the fact that primiparae rarely get accidental hæmorrhage. Again, it seems to be rather more commonly seen associated with intra-mural fibroid, that is to say, where the uterus is found to be nodular. Bright's disease is not infrequently associated with it, and it is supposed that it is commoner in cases where there has been a previous endometritis. Another thing which one almost always observes is that the patients are generally in a low state of health; it is not the robust, muscular, healthy sort of woman who gets this variety of trouble; it is the flabby and ill-conditioned.

The hæmorrhage which comes from underneath the placenta may be so small as to remain located under the placenta, and beyond detaching a few of the chorionic villi, and indirectly doing a little damage to the circulation of the foetus, and consequently retarding the aëration of its blood, it does not do much harm. It is often recognised

at full term by a patch of adherent placenta. But generally the blood makes its way from underneath the placenta between the chorion and decidua and shows itself externally.

A more serious variety is where it becomes *concealed*, that is to say, where it has stripped up the placenta and a good deal of membrane, but fails to quite reach the internal os. These two forms of hæmorrhage may co-exist. The prognosis of the former is not serious, but the concealed variety, if at all profuse, is a very grave condition indeed.

The *symptoms* of the ordinary accidental hæmorrhage, where blood is showing externally, is simply external hæmorrhage *plus* the fact that the woman is pregnant, and that perhaps some attempt is being made at labour. In the concealed form, however, the symptoms and physical signs are first those of shock and evidences of internal hæmorrhage, or of hæmorrhage which is not explained by the amount of hæmorrhage which is showing externally. Then there is a great deal of pain, not intermittent, like true labour "pains," but continuous pains, pains of tension, due to the sudden over-distension of the uterus with one to three pints of blood which has been infused into it. When one comes to palpate the abdomen, the legs are found drawn up, the abdomen is very hard and tender, the uterus is also very hard, perhaps irregular in shape (bulging in one direction more than in another), the outline of the foetus is obscured, no movements can be detected, and the foetal heart cannot be heard. And when one comes to examine per vaginam the head, which may have been presenting on a previous examination, may be pushed out of the way, and cannot be recognised by the examiner's finger.

"Accidental" hæmorrhage has to be diagnosed mainly from "unavoidable" hæmorrhage, and practically the distinction can only be made when you can pass your finger through the internal os, and can be sure that the placenta is not there.

The concealed variety of hæmorrhage has often led to many mistakes. I think the commonest mistake which students make nowadays, when they are taught so much about "tonic contraction of the uterus," is to mistake these two conditions. A woman is suffering from exhaustion and more or less shock, the uterus is felt to be very hard, the foetal outline cannot be felt, and the tendency is to

at once assume that there is some obstruction, and that it is a case of tonic contraction of the uterus. The distinction is that tonic contraction of the uterus occurs in cases of obstruction late in the second stage of labour, and accidental hæmorrhage which is concealed, practically always occurs before the membranes are ruptured in the antepartum or first stage of labour. Rupture of the uterus has also been mistaken for it, for this leads to shock and internal hæmorrhage; but there again rupture occurs in the second stage of labour and the physical signs are quite distinctive, because the uterus, if the foetus has escaped into the peritoneal cavity, is found to be small and contracted just as at the end of the third stage of labour, and the foetus is felt free in the abdomen outside. There are cases, too, where it has been mistaken for a secondary rupture of an extra-uterine gestation—a tubal gestation which has ruptured into the broad ligament, and at the end of so many months has ruptured again into the peritoneal cavity and led to an internal hæmorrhage with shock. But the physical signs are quite distinct, because one finds the foetus in the peritoneal cavity, with intra-pelvic fulness and displacement of an ordinary-sized or a slightly enlarged uterus.

The *prognosis* of most of these cases of accidental hæmorrhage is good if labour pains are present, but in the concealed variety it is bad. It is said that 50 per cent. of the mothers die and 90 per cent. of the children. The *treatment* of the ordinary form of accidental hæmorrhage, unless it is a very marked case of hæmorrhage, is to palliate—keep the patient quiet. If it is a multipara, and you are sure there is no obstruction, give ergot to try to induce uterine contractions, and at the same time put a binder on to apply equal pressure to the uterus. If hæmorrhage goes on, it is much better, especially after the twenty-eighth week, when the child is viable, to palliate no longer, but to rupture the membranes. The membranes are ruptured by the finger-nail made into a saw, or by a probe passed up along the finger. The almost immediate result is to make the uterus contract and retract, and so to close the uterine sinus, and then the hæmorrhage after this procedure almost always stops, and labour can be practically left to come on as it will.

In the concealed variety, however, mere rupture of the membrane does not bring on uterine con-

tractions, because the uterus is over-distended and is in a state of atony, and practically primary uterine inertia is present. It is therefore advisable, as the hæmorrhage is pent up, to try to release some of it; and you can generally do that, after digital dilatation of the cervix, by stripping off some of the membranes on one or other side, or all round if you like. Very often you can then free much of the blood-clot, which is often just above the internal os. This evacuation will give an opportunity for the uterus to contract down. If you find the hæmorrhage does not cease, but becomes apparent and continuous, it is much better to rupture the membranes and bring on labour as rapidly as

the hæmorrhage is concealed already, where there is very little uterine contraction, and the uterus wants emptying rapidly, there is no remedy so good as some form of hydrostatic dilator. Barnes' bags may be used, but require to be repeated. Remember that the greater the amount of manipulation the worse for the patient, owing to the danger of sepsis, and one therefore uses Champetier de Ribes' bag, which is made of waterproof silk. I show you one of them (Figs. 1 and 2). The size is so arranged that the maximum diameter at the top when distended is three and a half inches, so that one knows that when the bag has been expelled through the cervix, an ordinary foetal head



Fig. 1.



Fig. 2.

possible — practically empty the uterus. You will see that the treatment is just the opposite of that of the other variety. If it is necessary to empty the uterus quickly, the cervix must be dilated up rapidly by bougies, or more slowly by "bags." In England we do not use hydrostatic dilators, which remain *in utero*, in the apparent form of hæmorrhage, because one is afraid of turning an apparent hæmorrhage into a concealed variety. If we merely block the cervix and leave something in, it will take several hours to dilate, and as hæmorrhage may be going on all the time symptoms and signs of concealed hæmorrhage may ensue. In the concealed variety, however, where

will pass. It can be washed easily and is not distensible. It holds seventeen ounces of fluid. It is either filled with air or water, and well scrubbed and rendered aseptic. The cervix is dilated up to the size which will admit a thumb or two fingers, and the membranes are ruptured; then the bag is emptied again, rolled up into a small compass, then grasped between the two blades of specially prepared forceps and the whole passed up along the finger through the cervix. If you like you can draw the cervix down, but it is easy to pass it without. Withdraw the blades of the forceps one by one. Distend the bag with air, or preferably by an antiseptic fluid, for fear of it breaking, but

that is not very likely with this bag; Barnes' bags sometimes do break. The bag can be kept in until the cervix is dilated enough to allow it to come out by the uterine "pains," or it may be drawn out by the doctor, who can then, if need be, deliver the child readily, but if uterine inertia be present great caution must be taken to avoid post-partum hæmorrhage by trying hard to induce some uterine action. The only other thing to be careful of in these cases is that the child may need prompt attention, if still alive, owing to being more or less asphyxiated, from deficient aëration of its blood.

Now let us consider placenta prævia (unavoidable hæmorrhage). It is called "unavoidable" because it practically must take place anterior to or during the first stage of labour, during the spreading out or distension of the lower zone of the uterus. The definition generally given is that it is hæmorrhage due to the partial separation of a placenta which is attached to the lower zone of the uterus. Long ago Robert Barnes—I think in 1847—described the different zones of the uterus. He considered that the uterus could be divided into three zones. The fundal zone was that to which the placenta was ordinarily attached; the middle zone was that to which the placenta was usually attached in cases of accidental hæmorrhage (I did not mention this because it has not been verified); and the lower zone is that to which the placenta is attached when "prævia," and its upper limit is about one finger's length from the internal os at full term. This limiting line between the middle and lower zone of Barnes was called "the internal os" by Braune in 1872, the "ring of Bandl" in 1876, and the "contraction ring" by Schröder more lately. If the placenta or any part of it is attached to the lower zone there is almost sure to be hæmorrhage. We do not quite know why the placenta should get attached to the lower zone. There are two or three theories. The two main theories are—(1) That it is due to a low implantation of the ovum as a whole. That is a theory which Berry Hart has recently worked out. He says that in the ordinary way the ovum is attached only where the epithelium is denuded. He contends that that is the explanation why it is that the ovum, which may be impregnated in the tube, does not become attached on the mucous membrane of the tube habitually, but it finds its way into the

uterus, and, especially after the menstrual period, when there is no epithelium on the surface, becomes located somewhere on its lining membrane. He states that the reason of placenta prævia is that the ovum has not found any denuded patch until it reaches the lower zone, where perhaps some recent desquamative endometritis has been present. That has not been accepted by many writers; in fact, it is too recent a statement to have received much criticism. It rather bears out Lawson Tait's idea of the cause of tubal gestation being due to previous salpingitis, where the epithelium would be denuded. Another theory, which I think will turn out to be the right one, is that advanced by Hoffmeier. He believes that the placenta prævia is due to the development of the placenta within the decidua reflexa at the lower zone of the uterus. The ovum might have been implanted at the normal fundal end of the uterus, and yet the placenta had been developed from persistent chorionic villi at the other end. That has been accepted by a great many Continental writers—Martin, Altenbach, and Olshausen amongst others.

Neither do we know for certain why hæmorrhage should so often follow this low insertion of the placenta. There are many theories about it. Its exciting cause may be a blow, just as in accidental hæmorrhage. But the probability is that during the later months of pregnancy, long before any definite signs of labour come on, there is a distension and gradual gathering up of the lower zone into the main body of the uterus, and the placenta, being inelastic, is stripped off here and there, and therefore hæmorrhage results. Another theory, which has a great many adherents, is that the placenta, especially in the later months, grows more rapidly than the lower zone, and practically grows off the lower zone of the uterus, and the uterus becomes then detached from the placenta. In any case, however, the hæmorrhage is maternal, coming from the uterine sinuses, so that the child does not die from loss of blood *viâ* the foetal placenta, but from asphyxia.

The *symptoms* of placenta prævia are practically hæmorrhage in the later months of pregnancy. This hæmorrhage is not concealed, but is external, and can be diagnosed at once, and solely by passing the finger through the cervix; so that if one has a case of serious hæmorrhage in the later months

of pregnancy, in order to arrive at a diagnosis one *must*, as a step to diagnosis as well as treatment, get the finger through the cervix. That can be generally done by a little gentle digital pressure, without any dilating by bougies, especially in multipara. Occasionally the presence of the placenta can be recognised by vaginal palpation, obscured ballotement, &c., but these negative signs are not very reliable. The difficulty in laying down anything like definite *treatment* is the fact that no two cases ought to be treated on quite the same routine principle; so I want only to speak of the outlines of treatment. Some cases do well without being treated at all, but those are the cases where the hæmorrhage is slight and the labour "pains" are good. In such cases the presenting part is forced down through the internal os and compresses the placenta against the bleeding site underneath it, and so arrests the hæmorrhage. One often meets with cases of ante-partum hæmorrhages which have not been seen sufficiently early to make an ante-partum diagnosis, and yet one finds afterwards that the only hole in the membranes is adjacent to the edge of the placenta, instead of being practically at the opposite pole, showing it was a case of marginal or partial placenta prævia.

I did not mention the classes into which placenta prævia is divided. The most serious of all is the complete placenta prævia, where it is central, completely covering the internal os. Here the whole placenta is perhaps nine inches across instead of seven, and covers all the lower zone and part of the middle zone of the uterus. Then there is the partial placenta prævia, where it overlaps the os to a certain extent, but the membranes can be felt as well as the placenta. And then there is the marginal placenta prævia, where the membrane comes down to the os and can be felt, or where, if we do not examine until labour has begun, it feels like a fleshy tongue coming down into the cervix. Spontaneous deliveries would more often ensue were it not the case that in cases of placenta prævia malpresentations are more common, because the head does not adapt itself so well to the less cupped or otherwise altered lower zone of the uterus. The tendency, therefore, is to get a shoulder presentation which does not come down into the cervix, and does not compress the parts as a vertex or breech or thigh would do. Now,

suppose one has a case of hæmorrhage, and is satisfied that it is a case of placenta prævia by having passed the finger through the cervix. We will suppose that gestation has not progressed twenty-eight weeks, and that the child is not likely to be viable. In that case, if it is placenta prævia at all, it generally means that the placenta is a central implantation. The more centrally the placenta is attached to the lower zone the earlier does the hæmorrhage occur. It is obvious that it may be so, for it is attached over a larger surface of the lower zone. Therefore there is no particular reason why you should not bring on labour at once, and it is very much safer to do so. In cases occurring after the twenty-eighth week, if the child is dead there is no necessity to wait, and nothing can be gained by it. If it is alive, which you can perhaps detect by hearing the foetal heart or by movements, I still think that if the hæmorrhage is at all severe it is much better to interfere at once, so as to try and keep that child alive; otherwise the child is almost sure to die. It follows, therefore, that in any case of placenta prævia at any period, when one has once made the diagnosis, one ought, if the hæmorrhage is at all severe or has recurred, to consider that that woman, if allowed to go on with her pregnancy is doing so at the risk of her life, and it is much better to bring on labour at once.

As to the methods of treatment, I think the first thing to do when one gets a case of placenta prævia, where the diagnosis is sure, is to strip off a bit of the placenta from the lower zone by passing the finger through the internal os and sweeping it round. That will often enable that portion of the uterus which you have denuded to be drawn up by the uterine action, and so allow of further cervical dilatation. It also enables you to further dilate that part of the uterus with hydrostatic bags if you want to. If it is a case of marginal placenta prævia, it is very often quite sufficient to strip off the tongue of placental tissue from the lower zone of the uterus, then encourage the presenting part to come down, and leave the rest to Nature, and it is far better to do so if you can.

Supposing, however, that you find an undilated cervix and no pains—the most dangerous of all cases—it is better to strip off the placenta from that zone, and then to dilate it sufficiently to be

able to manipulate. You can dilate it with bougies if there is need for rapidity, or you can dilate with Barnes' bags, which may take perhaps twenty minutes or half an hour for each bag. The difficult cases are those where one has a rigid cervix in addition to these conditions, and it is extremely difficult to dilate a rigid cervix when there is placenta prævia. Having reached the membranes, easily done in a case of partial placenta prævia, and which one can generally do in the central varieties by working away in the direction of the thinnest part of the placenta, you then perform bipolar podalic version by the Braxton Hicks method, unless the cervix has been fully dilated by de Ribes' bag, and the vertex presents, when the forceps may be used. Turn the child by the bipolar method till you feel the knee, and then hook the knee down and bring it through the cervix to the child's half breech, which will compress the bleeding points far better than any hydrostatic bag. Labour pains will always come on, but perhaps not for an hour or two. There is not the least reason to draw the body of the child through. Neither the child nor the mother will suffer, and it gives you a chance of restoring the woman's vitality, which may be extremely low, by nourishment administered by mouth or rectum.

At the end of a certain number of hours the pains come on, the cervix yields still more, and the child may be helped through, or it may be left entirely to nature. I believe in many cases of placenta prævia which are treated up to that point satisfactorily, fatal injury is done by dragging the child through the cervix, causing laceration and subsequent sepsis, or inducing *post-partum* hæmorrhage. There is no bag which exerts better or more equal pressure over the whole of the lower zone than Champetier de Ribes' bag inserted right through the membranes, or, according to some, by pushing it right through even the central placenta. This is recommended, but I have never done it, and one wonders whether it is ever necessary, because one can almost certainly reach the edge of the placenta somewhere if it is stripped off sufficiently. The de Ribes forceps are quite strong enough to penetrate the placenta if need be.

Sometimes one is sent for to a case when one has not got the necessary instruments or assistants. You know it is a placenta prævia, but before inducing labour you want to get a nurse in the

house and you want a colleague to help you. In such a case it is advisable to tampon the vagina *and the cervix*, after sweeping the finger round, and if you can get antiseptic gauze, pass that up between the uterus and placenta through the cervix against the bleeding points, packing the vagina below. That almost certainly temporarily stops the hæmorrhage, and when you come again you find the cervix much more dilated. You come again a few hours afterwards and then you can dilate up with de Ribes' or Barnes' bag, perform bipolar version, bring down the leg, and leave the rest to nature. Roughly speaking that is the best routine practice.

The patient is always in a very low state from loss of blood, whether it be a mild or serious case, and the manipulations and perhaps the chloroform have pulled her down. Consequently it is necessary to be prepared to give her some form of rectal or venous infusion. The most rapidly effectual is venous infusion, but nearly as good results can be obtained by rectal infusion. I have several times had an opportunity of testing how rapidly injections are absorbed by the rectum. The other day I had a case, near this hospital, of ruptured tubal gestation. It was a five weeks' gestation, and the patient was apparently moribund. I opened the abdomen and found it full of blood, and detected a small nodule on the left Fallopian tube not bigger than a filbert. As there was no doubt that that was the point of rupture, I clamped it on each side and stopped the hæmorrhage, which one must always do before infusion, otherwise the patient will only bleed all the more when you have put more fluid into her. I then gave her rectal injections of brandy and water to the extent of three and a half pints. One could feel the pulse improving every second, both in quality and in bulk. In less than ten minutes the rectum was empty—the whole of the previous injection had been absorbed in ten minutes. That is not the first time one has found a similar thing. Sometimes the rectum refuses to hold the fluid at all, and then a venous infusion must be at once given. The best apparatus is Dr. Horrocks' infusion apparatus (Fig. 3). It is simply a little glass funnel to which is attached an ordinary india-rubber tube, and a glass nozzle, which is inserted into the vein. A silver instead of glass nozzle is perhaps better, as it is not breakable. All that has to be

done is to put a tourniquet round the arm high up, and this, even in the worst cases, will cause the median basilic or other large vein to dimly show itself. If that should not be so you can dissect down upon it. Then a couple of ligatures are passed underneath the vein, and the lower one



Fig. 3.

tied. A cut is made across the vein, and the nozzle with the water running from it is inserted into the vein, and the upper ligature tied over the vein and nozzle. Three to six pints are allowed to pass in, not very quickly, the speed being regulated by raising or lowering the funnel, so that no bubbles of air get in. A pint will easily run into the vein in three or four minutes. In half a minute you realise that the pulse is improving, and literally in two or three minutes the patient will tell you she feels all right again. Cut the vein right across and tie the upper ligature tightly. Sterilised water at 100° F. should be used, to which a teaspoonful of common salt has been added to each pint. The funnel should be kept full by water being constantly added.

[A demonstration of cases followed.]

THE DYNAMICS OF THE THORAX IN DISEASE.

A Clinical Lecture delivered at the Hospital for Consumption, &c., Brompton,

By **ROBERT MAGUIRE, M.D., F.R.C.P.,**

Physician to the Hospital, and Physician to Out-Patients, St. Mary's Hospital.

GENTLEMEN,—In spite of its alarmingly scientific title, my lecture will deal with matters of essentially practical interest, and I am led to give it by the remark of a medical man, who once told me, that for tapping a pleural effusion he always used the same trocar and cannula which served him for a similar condition in the abdomen. You will doubtless all perceive his mistake, but others may not, and such an ignorance of the conditions of intra-thoracic tension makes it probable that the more obscure relations of thoracic dynamics may be entirely unknown to many—important, as I think they are, for the proper knowledge of thoracic disease.

There is a marked difference between the tension inside the thorax and that inside the peritoneal cavity. It is true that the intra-abdominal tension varies. I heard Mr. Treves a little time ago, speaking of abdominal operations, say that he was never quite certain whether he would find positive or negative tension in the abdomen. Usually, however, the abdominal tension is positive. But in the thorax all the organs are under negative tension, and such a condition is a necessity for respiration, and desirable for other purposes. Dr. Harry Campbell, in his recent work on "Respiratory Exercises," says that the negative tension in the thorax is caused by the elasticity of the lungs, or, as he puts it, "pulmonary suction." But I am sure this is not the whole of the argument. An important factor in the causation of the negative tension is the anatomical fixation of the ribs and their cartilages, combined with their elasticity. The ribs are clamped down at their anterior and posterior extremities in a confined position, which their elasticity is ever trying to combat by forcing their anterior extremities outwards and forwards. This is not due to "suction" of the lungs, drawing the ends of the ribs inwards, for you may observe post mortem, in cases such

as I shall mention later, where carnification has destroyed completely the elasticity of the lungs, that the ribs spring outwards when their cartilages are divided. The position of the ribs is caused by anatomical development. Put broadly, the condition of the normal thorax may be stated thus :—the ribs are continually trying to expand outwards, while at the same time their inherent elasticity is continually compelling the lungs to contract towards their roots. These forces are in antagonism, the ribs trying to separate themselves from the lungs and the lungs from the ribs, and thus is caused the negative tension in the interior of the thorax. Such negative tension is greatly increased when the cavity of the thorax is expanded during inspiration, even to the extent of causing negative tension within the lungs themselves, this last being due to the fact that the orifice with which the lungs communicate with the outer air is so very small in comparison with the capacity of the lungs, that there is an interval of time before the pressure inside the lungs and that of the outer atmosphere can be nearly equalised. Even during expiration the negative tension in the lungs does not entirely disappear, but it can be changed into positive tension by voluntary forced efforts with a closed glottis or by involuntary cough.

Such are the conditions in health. The effects of variations of these in disease may be conveniently discussed as they are exerted upon—

- (a) The contents of the pleuræ.
- (b) The lungs themselves.
- (c) The other thoracic viscera.
- (d) The circulation of the blood.

The tension in the pleural cavity can easily be ascertained by the insertion of a tube communicating with a mercurial or water manometer, and that which exists in the presence of different varieties of pleural effusion is of considerable interest and importance.

The simplest form of pleural effusion is that of hydrothorax from the congestion which results from heart disease, &c. It is generally bilateral, is never great in amount, seldom necessitates removal, and has always a negative tension, that is, less than that of the external atmosphere. Similarly effusion from inflammation, though unilateral, has also a negative tension when its amount is moderate, and here, of course, is the error into which the medical man had fallen whom

I have recently quoted. It is because of this negative tension that we are obliged to use an aspirator, and not merely a trochar and canula for emptying the thorax of such an effusion. The negative tension of the effusion is associated with some curious phenomena. To some extent it helps to override the force of gravity. You would expect that the level of such a fluid would vary with the position of the body, and that it would be always a horizontal line. But it scarcely varies at all when the patient is moved, and not merely because of adhesions which may have formed. Moreover, it is not a horizontal line, but shows a distinct rise, of a dome shape, in the axillary region. As will be seen later, the lung collapses in the area covered by the fluid, and elastic traction is exerted by the other parts of the same lung; hence the fluid is, as it were, sucked upwards, and being hindered in its ascent on the inside by the root of the lung, rises to a higher level on the outside. Again, in serous pleural effusion of moderate amount, whether passive or inflammatory, the diaphragm is never depressed, as you would expect it to be, from the force of gravity by the weight of the fluid. One would, of course, estimate and detect the depression of the diaphragm by the position of the abdominal viscera. The stomach and liver are not lowered by an ordinary moderate pleural effusion, the intercostal spaces are not bulged, and the negative tension of the fluid is the cause of absence of these signs. The amount of the negative tension varies, but it may equal — 7 millimètres of mercury. But Dr. Calvert has shown that the amount of tension varies, as might indeed be expected, with the height in the fluid at which the puncture is made. At the lower part the tension may almost, if not quite equal that of the atmosphere, while, the higher we go, the lower is the tension. Thus, in spite of the generally prevailing negative tension inside the thorax, there is still a certain amount of pressure exerted by the force of gravity, the lower layers of fluid being pressed upon by the higher layers.

The tension inside the pleura in simple pleuritic effusion may sometimes be positive, that is, it may exceed the pressure of the atmosphere. But this only occurs in large effusions which entirely, or almost entirely, fill the pleural cavity, and are the result of an extreme degree of pleural inflammation. When this occurs we do get bulging

of the intercostal spaces and depression of the diaphragm.

But in purulent effusion we always find a positive tension, no matter how small the effusion may be, and hence arise the almost invariable depression of the diaphragm and bulging of the intercostal spaces, which are important points in the diagnosis of empyema. It has been urged as an explanation of these that the severity of the inflammation which leads to an empyema causes paralysis of the neighbouring muscles just as occurs with the intestinal muscles in peritonitis. But it is impossible to conceive of such an action on such powerful muscles as the diaphragm and intercostals, and a more ready and reasonable explanation is that the muscles are simply pressed down or outwards by the positive tension of the fluid.

We must inquire now, the reason of these differences of tension in different states of fluid effusion, in simple hydrothorax always negative, in pleuritic serous effusion usually negative, sometimes positive, and in purulent effusion always positive tension. We find it, I think, in the pathological processes which are severally involved in the production of the fluid. In simple hydrothorax we have a mere passive exudation, the result of an excess of pressure in the blood-vessels of the pleura, possibly combined with a hindrance to absorption from pressure on the lymphatics. The flow of fluid to the pleura is increased, and its rate of removal diminished. But this excess of pressure is very slight, the force leading to the exudation of the fluid can be easily overcome by pressure on the other side of the vessels. Consequently it is impossible for such an exudation to attain such a degree of positive tension as would entirely overcome the negative tension in the normal pleural cavity which is for ever tending to keep the lungs and the chest wall in apposition. A positive tension, too, would stop the exudation at once. But in pleuritic serous exudation we have added to this exuding force, the active force of secretion from the inflamed area, not great in amount, but still definite. Consequently a serous effusion from inflammation tends to be greater than one from simple passive congestion, and when the inflammation is severe and widely spread it may be so great as to entirely fill the pleural cavity, and, as we have seen, to

sometimes cause positive tension. A certain amount of positive tension is unable to overcome the conjoint forces of exudation and inflammatory secretion. Further, in purulent exudation we have another force added to these. As my friend Dr. Ewart put it to me, somewhat epigrammatically, it is the "inherent vitality of the leucocyte." The diapedesis of the leucocyte into the effusion which is characteristic of the formation of empyema is a very active process, and if we consider the enormous number of leucocytes exuded we must express the force exerted by them in the term of many foot-pounds of energy. Here we have a force which is capable of easily overcoming the natural tendency to negative tension existing in the pleural cavity, and its action is increased by the fact that most if not all empyemata, even in the early stage, are bound down by adhesions. They are more or less loculated, and hence the tension of the contained fluid will have a point against which it can act and so increase the positive pressure existing in the pus cavity.

When air gains access to the pleural cavity, its tension is never negative, thus differing markedly from that of serous effusion. Dr. Samuel West found that its tension varied from zero to + 9 millimètres of mercury. The presence of fluid in addition to air still further increases the positive tension. Such fluid is nearly always pus, and above you have seen the reason why this is always under positive tension. In valvular pneumothorax the positive tension is very great, simply because air is being constantly pumped into the cavity. Most of the openings into the pleura are more or less valvular at some stage, but when the opening is apparently not valvular at all there is more or less free communication with the atmosphere—a fact in itself enough to prevent any but the slightest negative tension; and moreover, the sudden access of air to the cavity, very different to the gradual effusion of fluid, immediately disturbs the approximation of the lung to the chest wall, and so air must be sucked in until all signs of negative tension disappear. The rapid advent of pus soon adds an additional force for the production of positive tension. The positive tension of a pneumothorax is an important matter in treatment, because of its injurious action on the movement and elastic traction of the lungs themselves, as will be described later, and its effects

are the more marked in local pneumothorax, as in a case under my care in the wards at the present time.

Let us now consider the variations of thoracic tension in regard to the lungs themselves.

Even yet one hears at times the expression, "the lung was compressed by fluid." But from what I have above shown, it is obvious that this can very rarely be the case. In the presence of any exudation in the pleural cavity, no matter what its tension may be, the first condition of the lung is one of collapse, from the unopposed action of the elasticity of the lung tending to draw its tissue towards its root. This will not remove the residual air from the lung, and doubtless Dr. Gee is right in saying that even the collapsed portion of the lung floats on the surface of the fluid exudation. The affected lung will also be drawn to the opposite side, a matter which will be more conveniently discussed later. After a time the collapsed portion of lung becomes absolutely devoid of air, infiltrated with young cells, and sometimes fibroid, as it is called, "carnified." It is true that you may have a certain amount of pressure exerted on the lung when the effusion is large or purulent, and its effect is illustrated when the lung begins to expand after the removal of only a small amount of the fluid which surrounds it. But it is impossible to get such a degree of positive pressure in the chest as will render the lung airless. You may place a piece of lung between two boards and jump upon them, without driving the air out of the lung. The state of lung in question seems to me to be a reversion to the embryonic type as the result of disease. The air is absorbed by the vessels. Embryonic cells are formed, and these may at times undergo a minor development into a lowly organised fibrous tissue at the expense of the special tissue. Such a process is common in other parts when vitality is lowered, but this is an abstruse point of pathology into which I must not enter here.

The tension of the air inside the air-passages and in various circumstances is undoubtedly a very important factor producing disease of the lungs. I need only mention the production of emphysema by increase of such tension, as was years ago worked out by Sir William Jenner and Mendelssohn, and I will go no further into the subject than to correct an error which has passed

from book to book as to the causation of emphysema by the playing of brass instruments. I do not know who is responsible for first putting forth this opinion, but he certainly did not play on a brass instrument himself. Nor do I, but I once managed to produce three notes from a post horn, and was astounded at the little effort required to obtain them. The fact is that the original observer was deceived by the grimaces of a trumpeter, evidences only of the efforts to tighten the lips, which are with this instrument the sound-producing parts, and which are kept tense by the efforts of the facial muscles and the pressure of the mouth-piece of the instrument. Doubtless you have all noticed the mark on a trumpeter's lips when he ceases playing, produced by this pressure. There is very little pressure in the chest during playing, not by any means so much as in singing, and indeed the performer is careful to prevent this, for otherwise he could not sustain the note. The puffed-out cheeks, seen in the players of larger instruments, are no evidence of volume of air employed, but are used as reservoirs, exactly as in using the blow-pipe. In my capacity of Honorary Physician to the Royal Society of Musicians, I have made inquiries from various players, amongst others from a veteran trumpeter, whose name is famed in the profession, and from an artist who plays what one may call the opposite instrument, the bass tuba, in the Wagnerian operas, and these gentlemen confirm the view mentioned. Again, one of my house-physicians informs me that he once played the cornet, and he, with his medical observation, tells the same tale. Further, the two gentlemen first named say that it is well known in the orchestra that the players on wind instruments do not suffer unduly from respiratory troubles, but are thought to enjoy better health than the others.

We have an effect of positive tension shown in the bronchial tubes in cases of bronchiectasis. This condition is, I think, an emphysema of the bronchial tubes, produced by the positive tension caused by cough, acting upon diseased and weakened bronchi, in every respect similar to emphysema of the lungs. There are, as you probably know, other explanations of this condition, but that given here seems to me the simplest and easiest.

Next we come to the effects of change

thoracic tension upon the other viscera in the chest, and practically this almost limits itself to the effects upon the heart. The heart is displaced by pleural effusion, but it cannot be too strongly insisted upon that this is not the effect of pressure. This was long ago demonstrated by Sir Richard Douglas-Powell, but is even now not universally understood. It is traction and not pressure which displaces the heart—traction by the opposite lung, which for ever trying by its elasticity to reach its own root, drags over to itself the mediastinum and its contents, now relieved from the opposing traction of the lung which has collapsed. It is true that this force may be assisted by the positive tension of a large quantity of serum, or of a moderate quantity of pus or air in the pleural cavity, or of a mediastinal tumour of any kind, but traction is always the main force concerned. Not only is the heart moved bodily towards the opposite side, but the position of its parts is altered. The base is the more fixed, the apex the more movable. Consequently the apex is the part the more displaced. Yet, little as the base is moved, the relations of the great vessels are sometimes disturbed, the pulmonary artery with its thin walls being the most easily affected, and consequently in such a condition we sometimes find a systolic murmur in or near the pulmonic area.

Again, the heart may be displaced by disease of the lung on the same side as the displacement, and this is said to be the result of traction by contracting fibrous adhesions, but I think such a view is somewhat incorrect. Such a displacement only occurs when there has been destruction of lung tissue, especially at the base of the lung, but there is always the accompaniment of pleural adhesions. There is always loss of volume, for though a cavity may form, taking the place of destroyed tissue, yet its contained air will be frequently driven out by cough and incompletely renewed by the deficient inspiratory expansion. The chest wall gives way to some extent, but obviously there must be an increase of the normal tendency to negative tension, incapable of being balanced by expansion of the disabled lung. The result is what one may call suction of the mediastinum, and with it of the heart and of a portion of the healthy lung, towards the diseased side.

The last part of my subject, the influence of

variations of thoracic tension upon the circulation of blood and also of the lymph, is, I think, very important from a practical point of view, and I place in the first rank the consideration of a physical sign which has almost ceased to be mentioned in the books. This is the *pulsus paradoxus*, the condition in which there is no pulse—that is to say, where the pulse ceases to be felt at the wrist during inspiration. It was once thought to be a definite guide to the diagnosis of pericardial adhesions, and the explanation given of its occurrence was the following. It was believed that when adhesions existed between the pericardium, the large vessels, and the chest wall, the upward and outward movement of the chest wall in inspiration caused the adhesions to pull on the aorta to such an extent as to obstruct its lumen and prevent the left ventricle driving blood into it; consequently no pulse could reach the wrist. On the face of it it is difficult to conceive that such a strong-walled vessel as the aorta could be so obstructed, and in practice it was found that the sign was not only absent in many such cases, but was found in many other conditions. For this reason no importance whatever has, for many years, been attached to the sign, and at the discussion on Adherent Pericardium held at the Medical Society of London during its last session, the phenomenon was only mentioned once, and then to be put on one side as useless. But I think it is far from useless, and, on the contrary, a very valuable indication of weakness of the left ventricle of the heart. It is essential, however, to understand its true mechanism, and that, I venture to think, is given in a small article of my own on the "Pulse" in Fowler's 'Dictionary of Practical Medicine.' Let me explain it again here. The heart, like all the other organs of the thorax, performs its work, surrounded by the there-prevailing negative tension. The negative tension, even in the normal state, hinders the heart, theoretically, in its effort to drive the blood out of its cavities, and the hindrance must be supposed to be the greater during inspiration; yet a normal left ventricle is so strong that even the increase of negative tension during inspiration cannot produce any effect on the pulse as perceived by the finger. But let the inspiratory effort, and therefore the negative tension, be very great, or let the left ventricle be weak, or *a fortiori* let these two conditions

be combined, and the negative tension will be too much for the ventricle, and no pulse will reach the wrist. A combination of these two conditions is found in the dying, who gasp in inspiration, when the left ventricle is at its weakest, and then you will very frequently find the *pulsus paradoxus*. Similarly, at times, I can produce the phenomenon on myself, especially in the middle of the forenoon, when one's circulation is feeble, and perhaps I may be able to do so now. I stand perfectly erect, with the shoulders thrown backwards as far as possible, so as to increase the inspiratory capacity of the chest, the arms fully extended, crossed behind my back, and I will ask one of you to feel my pulse while I take a rapid deep inspiration. [Several of the audience here perceived that the radial pulse became imperceptible.] Now this same phenomenon is of value in the detection of disease. A few weeks ago a medical man consulted me about an irregular and turbulent action of his heart, and the diagnosis of his case lay between gout and heart weakness following influenza. I observed that as he sat in the chair breathing quietly his pulse disappeared with each inspiration, and this sign helped to guide me to the correct view that he was really suffering from the effects of influenza. Suitable treatment has now caused disappearance of all his symptoms. I feel certain that many of the pulses described as "intermitting every third, fourth, or fifth beat," are really instances of *pulsus paradoxus*. Further, if a patient in whom you suspect cardiac weakness be asked to take a deep inspiration, the observance of this sign will materially aid your diagnosis in an early stage of the disorder.

Again, the negative tension of the thorax, or rather its deficiency, is a cause of some forms of dyspnoea. Perhaps you have noticed that thickening of the pleura, especially when tuberculous in origin, is accompanied by much panting, always on slight exertion, sometimes even when the patient is at rest, and this when the lung is only slightly, if at all, involved. In a former lecture I have told you that one cause of the dyspnoea of emphysema is the stretching of the blood-vessels of the lung, which throws strain on the right heart; but defect of negative tension is an additional cause, and acts in the following way. The normally negative tension of the interior of the thorax, especially when increased by inspiration, is a

most important agent in the mechanism of the circulation. Doubtless it has a powerful influence in producing the diastole of the ventricles, but especially does it act by drawing blood from the large veins to the auricles. Now if the range of inspiratory movement be diminished, either by pleural adhesions or by emphysema, there will be a less than normal amount of suction of blood into the auricles, a lessened supply of blood to the lungs, and consequently dyspnoea. In fact, the lung surface available for respiration may be quite sufficient, but the amount of blood supplied for oxygenation will be deficient.

NOTES, ETC.

On Scrofulosis. Ritter ('Allg. Med. Central Ztg.,' 1897, lxvi. 654).—Does scrofulosis tissue contain tubercle bacilli at the outset? This question Ritter attempts to answer by a series of experiments. The result is given by him before the Berliner Medicinische Gesellschaft (1897, vi, 23).

In the first place, the bodies of nineteen scrofulous children, who had died from intercurrent diseases, were dissected; about a dozen sections were taken from each enlarged lymphatic gland and stained for tubercle bacilli, and portions of the bronchial, mesenteric, and cervical glands were injected into the abdominal cavity of guinea-pigs. Only in one single case, that of a child who died from whooping-cough, were bacilli found, and of these only six in fifty sections. There was also present a caseous pneumonia with a moderate accumulation of tubercle bacilli. Further experimentation was made upon living scrofulous children. In 129 cases of eczema, neither staining nor culture showed the presence of the tubercle bacillus in the contents of the vesicles.

Animal experimentation, which was undertaken in thirty-four cases, also resulted negatively. Forty curetted and fifteen extirpated cervical glands were next examined; in only two cases in which there already existed very great changes in the bones, could tubercle bacilli be demonstrated. The same result followed experiments in animal inoculation. Finally in twenty-three children suffering from

multiple suppurative processes of connective tissue, examination of the pus gave a negative result.

From this we may probably conclude that scrofulosis and tuberculosis are not identical, and that the tubercle bacilli are not to be regarded as the instigators of the former. We should ascribe to these bacilli only a secondary role in these cases, and this even if they be found in every case of advanced scrofulosis. The hypothesis of the formation of spores by the tubercle bacilli in scrofulosis is not tenable. We are no more justified in designating as tuberculosis those cases in which single tubercle bacilli are found, than we are in declaring a nurse to be suffering from diphtheria, because we find that there are a few diphtheria bacilli in her oral cavity. That one disease by the changes wrought may easily open an entrance for the excitors of another disease is shown, for example, by the invasion of the streptococci in cases of diphtheria; and it is certain that tuberculosis may be developed on the soil of a scrofulosis, that a slow intermingling may occur, and that there exists a great affinity between the two diseases.—*Pediatrics*, July, 1898.

The Uses and Dose of Apomorphine.—Dr. Robert H. Babcock ('American Medico-Surgical Bulletin,' June 10th) corrects from his own intimate personal knowledge of this drug many erroneous impressions current concerning it. Patients, he says, can tolerate very much larger doses than is commonly supposed, and by the mouth as much as two grains at a single dose may be given without nauseating. It is more apt to cause emesis when taken in the morning before breakfast.

The combination of a small dose of apomorphine, however, with other nauseating expectorants increases their efficacy. Dr. Babcock says that its effects are so satisfactory and it is so easily administered in pill or capsule, when for any reason it is not desirable to prescribe a syrup, that it has become his favourite remedy and main reliance in the treatment of both acute and chronic bronchitis. Combined with codeine or morphine, troublesome cough can be allayed without at the same time arresting bronchial secretion; indeed, the sputum will be increased, while at the same time the cough is moderated in violence and frequency.

The author's usual dose, administered by the mouth, appears to be about a fifth or a quarter of

a grain, up to one half, and sometimes to one grain, every three or four hours. In the case of a nursing baby a sixtieth of a grain every four hours in syrup of wild cherry bark was given with great effect in constant dry cough. The author calls special attention to the necessity of obtaining pure apomorphine (and recommends Merck's), as he has seen the soporific and other effects of morphine induced by impure specimens. It must not be prescribed in mixture with potassium iodide.—*New York Medical Journal*, July 2, 1898.

A LITTLE startling in the line of therapeutics is the suggestion of Dr. Jacob, of Leyden's clinic, in Berlin. He proposes in disease of the central nervous system to inject drugs directly into the subdural space. The absolute security with which a lumbar puncture may now be done indicated that some further step in this line would come. The Quincke puncture, though expected to be of great use in therapeutics, has only proved of service in meningitis serosa, and simple meningitis not due to bacteria, though there are some wonderful cures reported in extremely threatening cases. Jacob has been able to inject 150 cubic centimetres of various solutions of drugs into the spinal canal of animals without causing symptoms. In two cases in human beings a solution of iodide of potassium has been so injected without any untoward results, though the cases were too far gone to hope for anything but passing amelioration of symptoms.—*Therapeutic Gazette*, June, 1898.

Treatment of Uterine Tumours.—Drs. Hartmann and Fredet describe five cases of uterine fibroids in which recourse was had to vaginal ligation of the base of the broad ligaments. The immediate results were quite satisfactory.

Post-Graduate, July, 1898.

By a new process of sterilisation Nestlé has succeeded in producing an unsweetened condensed milk not only free from bacteria but retaining all the essential qualities of fresh milk. Those responsible for the welfare of invalids and children will especially appreciate the practical value of having at easy command this reliable preparation, called by Nestlé the Viking Brand, one part of which mixed with three times the quantity of water produces a pure good milk.

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* Specially reported for The Clinical Journal. Revised by the Author.

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A CLINICAL LECTURE

ON

CEREBRAL LOCALISATION.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, London, January 11th, 1898,

BY

CHARLES E. BEEVOR, M.D.Lond., F.R.C.P.,
Physician to the Hospital, and to the Great Northern Central Hospital.

GENTLEMEN,—I propose this afternoon to first of all go rapidly over the anatomy of the cortex, and afterwards to speak about the functions of it, subsequently showing one or two cases to illustrate the teaching.

First, then, I will enumerate the convolutions, and indicate them on the diagrams which you will see before you; and I shall endeavour at the same time to point out the easiest way that I know of to understand the convolutions and their position. First of all I show you a model of one hemisphere by itself, that is, without the pons, crura, or cerebellum. You are, of course, aware that the two hemispheres are joined together by the transverse commissures, of which the corpus callosum is the most important. The diagrams before you represent the brains of a Macaque monkey, the brain of an orang-outang, and the human brain. The diagram of the orang-outang brain is tilted forwards, so that you may see as much as possible of the part which is above the fissure of Sylvius.

We will now refer to the fissures. It used to be taught that the fissures were different to the sulci, that the fissures divided the cortex into lobes, and that these lobes were further subdivided by sulci into convolutions. But sometimes the fissures and sulci are described as the same thing. Nevertheless I think it helps one if the fissures are kept separately from the sulci, making the great division into lobes by fissures and subdividing the lobes into convolutions by means of the sulci.

You will notice that on the outer surface of the

brain there are three chief fissures. The fissure of Sylvius is a very long and distinct one, and is very easy to find out, starting as it does from the base of the brain and winding to the outer surface of the hemisphere. Next is the fissure of Rolando, which is the best point to start with in examining the convolutions of the brain; it is better not to begin with the frontal convolutions. The fissure of Rolando is found by running your eye along the median surface of the brain from before back, and it is the first fissure to be described, which starting from the middle line can, at first almost at a right angle, be traced downwards and forwards almost as far as the fissure of Sylvius. It is well marked and is easy to find, as I have shown you. The general direction of the fissure of Rolando forms an angle of 67° with the middle line. Of these fissures the last one is the parieto-occipital fissure, which is very small indeed in man on the outer surface, and small in the orang-outang, but in the monkey it is much larger; it is prolonged on to the median surface, where it joins the calcarine fissure.

The fissure of Sylvius separates the brain into the following lobes: the temporo-sphenoidal below, from the parietal and the frontal above it. The fissure of Rolando separates the frontal lobe in front from the parietal lobe behind, and the external parieto-occipital fissure separates the parietal from the occipital.

There are three chief sulci—(1) the præcentral sulcus, (2) the intra-parietal, and (3) the parallel. The præcentral sulcus is in front of and parallel to the fissure of Rolando, and is divided into a superior and an inferior part. The intra-parietal sulcus is within the parietal lobe, and is behind the Rolandic fissure and at first parallel to it, but it afterwards winds backwards and gets parallel to the median edge of the hemisphere. The parallel sulcus is thus called because it runs parallel to and below the fissure of Sylvius. This sulcus in the monkey is a very important one, and it runs further back than the fissure of Sylvius. I show you these sulci in a human brain which has been hardened in carbolic acid. We have thus three fissures and three sulci, which I have mentioned.

We will now give some attention to the convolutions which are separated from each other by these fissures and sulci. The best way is to take the fissure of Rolando and notice the convolutions

on either side of it. These are the ascending frontal and the ascending parietal. The ascending frontal convolution is bounded in front by the præcentral sulcus, mentioned just now, and the ascending parietal is bounded behind by the intra-parietal sulcus. This shows the use of remembering these sulci, for they enable one to fix the limitations of the ascending frontal and parietal convolutions. In front of the ascending frontal convolution we have three frontal convolutions having a horizontal direction, and behind the ascending parietal convolution there are also three, namely, the parietal lobule, the supra-marginal convolution, and the angular convolution. The parietal lobule is the continuation backwards of the upper end of the ascending parietal convolution, and is separated by the intra-parietal sulcus from the next convolutions. The supra-marginal and the angular convolutions are difficult to follow, because they differ in different brains. The best way is to find the fissure of Sylvius and trace it to its posterior end. The convolution which is round the posterior end of the fissure of Sylvius is the supra-marginal. Next trace the parallel sulcus backwards, and the convolution round the end of this is the angular. This is better seen in the monkey, while in the orang-outang it is not so easy to find out.

There are three other convolutions in the occipital region which I will not trouble you with, because they are comparatively unimportant for our present purpose.

Now if we take the mesial surface of the brain, we have also there certain fissures, sulci, and convolutions. There are three fissures which we must note in this area, besides the internal parieto-occipital which we have mentioned in connection with the external, viz. the calcarine, joining the parieto-occipito, the collateral, and the callosomarginal. The calcarine fissure begins at the posterior pole of the hemisphere, and arches upwards to meet the internal part of the parieto-occipital fissure, and then downwards and forwards to end below the posterior part of the corpus callosum. Below the calcarine, which is a very important fissure and is well marked in the brains of small monkeys where even the fissure of Rolando is absent, is the collateral fissure having a horizontal direction. The callosomarginal begins at the median edge of the hemisphere just behind the

upper end of the Rolandic fissure, and runs downwards and then forwards round the anterior end of the corpus callosum.

There are six convolutions here to remember:—The marginal belonging to the frontal lobe, between the median line and the calloso-marginal fissure, and extending from behind the level of the Rolandic fissure forwards and downwards to the commencement of the Sylvian fissure at the base of the brain; the cuneus, the wedge-shaped convolution between the calcarine and the parieto-occipital fissures, and belonging to the occipital lobe; the quadrate, the square convolution in front of the last fissure, forming the continuation of the parietal lobule on to the mesial surface; the lingualis, a convolution below the calcarine; the hippocampal lobule at the extreme anterior end of the temporal lobe, to which lobe both of these convolutions are now considered to belong ('Quain's Anatomy,' tenth edition), and lastly the gyrus fornicatus winding round the corpus callosum from the front, and extending posteriorly into the hippocampal convolution.

Taking the brain in the way I have done, beginning with the fissure of Rolando, and then finding the ascending frontal and parietal convolutions on either side of it, you will find if you exclude these two that everything goes in threes, and bearing this in mind helps you very considerably. There are three frontal, three parietal, three occipital, and three temporo-sphenoidal convolutions; three chief fissures, and three chief sulci on the outer surface, while on the median surface there are three fissures and six convolutions.

Now we will deal with the functions of the cortex cerebri. The best way is to divide the cortex into the excitable part and the non-excitable part. By excitable I mean the regions which respond to excitation by a faradic current. If you place the two electrodes connected with the secondary coil of a battery on the brain and excite the cortex, you get movements in different parts of the body when they are applied over certain areas, but not when they are over others. The excitability of different parts of the brain in the macaque monkey, and in the orang-outang has been examined in detail by Mr. Horsley and myself. The excitable region of the cortex in the monkey includes the ascending frontal, the ascending parietal, the parietal lobule, and the posterior half of the first, second, and

third frontal convolutions. The part in front of that region is not excitable, neither is the part behind it, including the temporo-sphenoidal lobe, or at any rate, this is excitable in only a very slight degree. On the median surface it was found by Horsley and Schäfer that you get movements by excitation of the marginal convolution, and especially of the trunk and lower limbs. The anterior part of the cortex, which is unexcitable, has been supposed to be the seat of the highest mental process. Whether that is or is not the case one cannot say for certain, but there is considerable evidence in favour of this view.

Well, what are the movements one gets by stimulation of the excitable part of the cortex? I must confine myself in the short time at my disposal to giving you some results arrived at by myself in collaboration with Mr. Horsley. We examined every twelfth of an inch of the motor cortex by making an accurate drawing of the part of the cortex under observation on paper ruled by lines which divided it into squares of this diameter, and then each square was stimulated, and the resulting movement recorded. The question arises, how is one to remember and learn these movements and their localisation in the cortex? I think the best way is to begin on the marginal convolution on the median surface, and to go over to the outer surface following down the fissure of Rolando, and the ascending frontal and parietal convolutions on either side of it to the fissure of Sylvius, and then to come forwards so as to end up in the frontal convolutions. If you do that you will find that the movements are represented in the following order: on the median surface you get the movements of the trunk: next you have at the upper end of the Rolandic fissure the movements of the lower limb, then of the upper limb about the middle of the fissure, and of the face at the lower end, *i.e.* the order from above down is trunk, leg, arm, face; then going still further forwards you get movements first of the pharynx, and then of the larynx including the vocal cords (Horsley and Semon), the latter being just behind the posterior part of the third frontal convolution. The order I have given you is a gradation from the lowest to the highest functions, that is to say, from the movements of the trunk, and of the extremities to those of the face, and of the vocal

cords, which are used for speech, the most highly evolved function, and only found in man.

In the lower and upper limb representations you can subdivide the movements still further. Mr. Horsley and I took each of these squares, and worked out the representation of the movements of the different joints, finding out in what order they were moved by stimulation of each of the small areas we had mapped out. We got different results according to whether our subject was the monkey or the orang-outang. The order in which we found the centres arranged in the ascending frontal and parietal convolutions in the orang-outang is probably that in which they occur in man, namely, from above downwards along the Rolandic fissure, in the order of hallux, small toes, ankle, knee, hip, and for the upper limb, in the order of shoulder, elbow, wrist, fingers, and thumb. In the face area at the lower end of the ascending frontal and parietal convolutions movements are obtained of closing the opposite eyelids, movements of elevation of the upper lip, and of retraction of the angle of the mouth; movements of the tongue are represented by a very large area. Then there are movements of mastication—very curious rhythmical to-and-fro movements of the lower jaw—which were present the whole time the electrodes were left on, and the movements of swallowing, and of the soft palate, and of the larynx.

A movement I have not yet mentioned, so as not to confuse your minds, is the movement of the head and eyes to the opposite side. This is chiefly in the posterior part of the three frontal convolutions, but most in the second frontal convolution just in the angle of the præcentral sulcus; above this part you get movement of the head only, while below you get movements of the eyes chiefly. The movements which we obtained in the eyes were parallel, and to the opposite side; the head also turned to the opposite side, and in so doing brought into action the sterno-mastoid; so that the left sterno-mastoid was thrown into action by stimulating the cortex on the *same* side, and the head was turned with the face to the right. The reason of this is that the movement was required to be to the opposite side; it is immaterial whether the muscle moved was on the same side, or on the opposite side; the brain knows nothing about muscles, only of movements,

and it is a mechanical convenience that the left sterno-mastoid should be used to turn the head to the right side. I believe there will be no difficulty in understanding how it is that the head turns to the opposite side from the action of a muscle on the same side as the cortex stimulated, or that it is common to have paralysis of the left sterno-mastoid in right hemiplegia in the early comatose state.

I wish to call special attention to the localisation of the movements of the thumb and of the big toe, because they are the spots at which fits often start. The representation for the big toe is at the upper end of the fissure of Rolando, near the middle line. I show you also the position of the representation of the thumb; you will see it is close by this bend in the fissure of Rolando, called the genu, which occurs also in the monkey and the orang-outang very markedly indeed. Just above that bend, in the ascending and parietal convolutions, is the localisation for the movements of the thumb.

Now, in mapping out these areas I wish you to understand that there is no hard and fast delimitation. One can find squares, irritation of which produce disturbances in more than one limb. For instance, you may produce movement of the shoulder at one spot, and of the leg at another near to the former, but there is a certain place between the two, stimulation of which causes movement of both leg and shoulder. All these movements are unilateral—that is, if you stimulate one half of the brain you get movements on the opposite side only. But there are also certain movements which are bilateral. The chief of these latter are the movements for opening and closing the mouth, and for mastication and swallowing, as well as some movements of the tongue and of the vocal cords; that is to say, if you stimulate one side of the cortex you get the muscles on both sides thrown into action.

One set of movements are difficult to understand, and they are very interesting indeed. It was formerly taught that the movements of the tongue were like those of the arms—that is to say, if you stimulated the left cortex the right half of the tongue only would move, and if you stimulated the right half the left would move. But that is not correct; you cannot get one half only of the tongue to move by stimulating one side of the cortex. The chief movements of the tongue

which we obtained were, advancing straight, protrusion of the tip to the opposite side, and lastly the rolling of the dorsum of the tongue to the cheek on the same side, which comes inwards to meet it—all these are localised in the lower part of the ascending frontal and parietal. In the front part of the lower end of the ascending frontal is the representation of the movement for advancing the tongue, the back part is for retraction, while the part just in front of the lower end of the fissure of Rolando is for the rolling-over movement. The whole of the part I show you, up to the level of the top of the pre-central sulcus, is for the movements of the protrusion to the opposite side. The movement is very curious in the tongue, because in protrusion to the opposite side you find that if you stimulate the left cortex, after having divided the tongue vertically along the raphé into two equal portions (under an anæsthetic, of course), the half of the tongue on the same (left) side comes forwards, and the opposite (right) half goes backwards, producing a see-saw movement. When the tongue is not divided one half pulls backwards and the other forwards, the resultant being a protrusion of the organ, with the tip directed to the opposite (right) side.

In the cortex itself there are three functions. It is a subject which has been observed by Mr. Horsley, and I have had some cases which have shown the same results. First there is the motor function, by which I mean that if you stimulate the motor cortex you get movements of some part of the body. Secondly, there is the muscular sense. This muscular sense is lost in that part of the body represented in the part of the cortex removed; that is to say, if you remove the leg centre in the cortex you will get paralysis of the leg on the opposite side, and at the same time you will get loss of muscular sense in the joints of that limb. Thirdly, there is slight loss of tactile sensibility. The touch often feels like a touch to the patient, but not always; but the patient cannot localise it, and usually puts his finger an inch or two from the spot. The three symptoms produced by removal of a part of the cortex are therefore—(1) loss of power corresponding to the part removed, (2) loss of muscular sense, and (3) some loss of tactile sensibility.

I have not said anything about the pathology of the cortex, but I may mention the symptoms

which are produced by lesions of the motor cortex.

The first result of a lesion such as a tumour is irritation, which produces localised fits; and the second is destruction of the cortex, causing local paralysis. Therefore, in the case of a growth of the cortex, you first of all have fits consequent on the irritation, and then, as the growth increases and produces destruction of the cortex, you get paralysis of the part supplied by that particular portion of cortex. These fits are epileptiform in character, as in the case I will show you presently, and are local in distribution, and are often without unconsciousness.

Now a word or two about the localisation of sensation in the cortex, in addition to what has been said about the motor cortex. This is a very difficult question indeed, and consequently opinions differ very widely as to the parts of the cortex in which sensation is represented. According to the experiments of Horsley and Schäfer, in which the gyrus fornicatus, — the convolution which runs round the corpus callosum on the median surface of the hemisphere, and extends backwards and downwards into the gyrus hippocampi—has been removed in the lower animals, anæsthesia was produced on the opposite side, but it was not possible to find any definite part producing anæsthesia of the arm or leg alone. Of course this part is very difficult to get at; you have to separate the hemispheres to remove part of this convolution, and it is very difficult to remove just the particular part you want to. Still their operations did undoubtedly cause loss of tactile sensibility and also of pain sensations on the opposite side of the body. Others consider that sensation is localised in the parietal regions. I have had a case of abscess in the parietal region in a boy who was kicked by a horse, who was operated on by Mr. Horsley, but I could never discover that he had any anæsthesia from the lesion there, so that I rather doubt that the parietal region contains the localisation for sensation.

Of the special senses sight is localised mostly in the occipital region, the two chief convolutions for this localisation being on either side of the calcarine fissure; above it is the cuneus or wedge shaped convolution, and below the calcarine you have the lingual convolution. If one occipital region is removed, you get loss

of vision in each eye for the opposite half of the field of vision. The angular gyrus is also said by some observers to give rise to what is known as "crossed amblyopia," but that is rather doubtful; it means a diminished field of vision of the periphery all round up to 5° in one eye (that opposite to the side of lesion), and 20° in the other eye, together with great diminution in the acuity of vision. The angular gyrus on the left side is the situation for what is known as "word blindness;" a form of aphasia where the patients are unable to understand visual communications. Hearing is localised in the first temporo-sphenoidal convolution according to the experiments by Ferrier, but it has been contested by Schäfer. "Word deafness," or an inability to understand auditory communications, is produced by lesions in the temporo-sphenoidal convolutions at the posterior part. The sense of smell is localised at the extreme anterior end of the temporo-sphenoidal lobe in what is known as the hippocampal lobule. I had a case with Dr. Jackson some years ago, which was published in 'Brain,' where the woman had curious fits, which began with the sensation of a peculiar smell, which smell she always experienced before the fits. She died, and we found there was a growth in the extreme anterior part of the temporo-sphenoidal lobe. That this is the location for smell has also been found by experiments on monkeys by Ferrier.

Now we will give some attention to the internal capsule. The internal capsule is the part within the brain in which the fibres of the brain pass up and down between the crus cerebri and all parts of the cortex, and it is situated between the basal ganglia, and consists of white fibres. I show you the situation in a brain which has been cut across horizontally. You see the lenticular nucleus, and the caudate nucleus, and the optic thalamus, between which is the white part known as the internal capsule. If you stimulate by the faradic current the cut fibres of the internal capsule, as Mr. Horsley and I did, you find you get a certain part excitable, and certain parts in front and behind of this part non-excitable. The fibres of the internal capsule have the same functions as the part of the cortex with which they are connected. Hence the excitable part of the capsule connected with the motor cortex is in the middle, and the non-excitable parts are in front and

behind this part. The first movements you get, when stimulating gradually from the anterior part which is not excitable to that which is, are opening of the eyes, then turning the eyes to the opposite side; then you get movements of the mouth opening, head turning, movements of the tongue, retraction of the angle of the mouth; then movements of the shoulder, elbow, wrist, fingers, and thumb; then of the abdominal muscles, and you can get movement of the rectus abdominis of the opposite side only by itself; lastly we obtain movements of the hip, ankle, knee, hallux, and toes. The excitable part occupies in a horizontal section, taken through the middle of the vertical height of the capsule, the posterior one sixth of the anterior limb and the anterior two thirds of the posterior limb. The fibres of the anterior non-excitable part come from the frontal part of the cortex. Behind the excitable part is the non-excitable area, which is considered to be for the conveyance of tactile sensibility; that is to say, if you get a lesion there you get anæsthesia on the opposite half of the body. From the optic thalamus you have fibres which are known as the optic radiations, and which go to the cuneus and lingualis. A lesion of these fibres would cause hemianopia on the opposite side.

The internal capsule is the place, *par excellence*, for hemiplegia. If the lesion affects excitable fibres, you have motor hemiplegia, and if further back you have sensory trouble as well, and if further back still you have hemianopia. Some students experience a difficulty in remembering what form of hemianopia you get from a lesion here, but it is easy to follow. With a lesion of the left internal capsule you have motion paralysed on the right of the body, and if there is loss of sensation you get loss of sensation on the right, and if you have an affection of vision the man cannot see objects on his right side with either eye, so that all three go together. An extensive lesion, therefore, of the left internal capsule gives loss of power on the right side, anæsthesia on the right side, and inability to see objects in the right half of the field of vision. If you remember that, when a man loses the power of his right hand, and also from the same lesion loses the power of seeing, it is in his right field where the right hand works that the defect occurs, you will have no difficulty in remembering the

association. Hearing, and sometimes taste and smell on the opposite side, are also occasionally lost from a lesion of this part.

[A case of epileptiform attacks beginning in the foot to illustrate cortical localisation was shown, and also one of hemiplegia, hemianæsthesia, and hemianopia as an example of lesion of the internal capsule.]

DR. HAGGARD concludes a communication on "Ectopic Gestation" with the following recommendations:—1. In unruptured ectopic gestation the vaginal operation, if congenial to the surgeon, may be elected. 2. In non-active cases of encysted hæmatocele vaginal section and drainage is the operation of choice. 3. The situation of the mass low down, and the broad, roomy vagina of parous women are favourable to the lower route. 4. Before evacuating ectopic collections *per vaginam* preparation for abdominal section should be made. 5. In free or uncontrollable hæmorrhage, after removing the products of ectopic gestation vaginally, the abdomen should be opened at once. 6. When abdominal section is necessary after colpotomy the preliminary vaginal incision (*a*) will confirm the diagnosis; (*b*) facilitate the abdominal work by removing clots through the vagina instead of through the abdomen; and (*c*) establish an efficient avenue for drainage. 7. The vaginal operation in appropriate cases is attended with less mortality.—*American Gynecological and Obstetrical Journal*, July, 1898.

Infection with Pneumococci.—Mr. Ballay and A. Halipré state that children frequently suffer from infection with pneumococci. Among the clinical manifestations which result, those affecting the larynx are most interesting and important, and consist of tirage and attacks of suffocation. Sometimes there are false membranes upon the vocal cords, so that the expression *pneumococcus croup* is justified; in other cases the vocal cords are unaffected, and there is no lesion of the larynx, the affection being probably a spasm of the glottis. The authors report two cases. One of them closely resembled diphtheria, but a bacterial examination proved the true nature of the disease. Intubation was successfully performed, with good results. In the second case broncho-pneumonia was present. Intubation gave great and immediate relief, but the child died in spite of all efforts to save it. In such cases the authors recommend cold packs of the thorax, subcutaneous injections of caffeine and artificial serum, and in the event of otitis media instillations of oxygenated waters in the external auditory canal.

American Journal of Obstetrics, July, 1898.

A CLINICAL DEMONSTRATION

ON A CASE OF

TUMOUR IN EACH GROIN,

At St. Mary's Hospital.

By EDMUND OWEN, F.R.C.S.

GENTLEMEN,—This young woman is a housemaid who, until a few weeks ago, was bravely struggling through her work in a country situation. She was sent up to me by Dr. Sydney Austin, of Lingfield, who told me that he was sure that she would prove of great clinical interest to our class. All of you in the front row have had the opportunity of quietly and leisurely examining her in the adjacent room, and the great variety in the opinions which you tell me you have formed shows that Dr. Austin's estimate of the clinical value of the case was by no means over-stated.

The tumour upon the right side is about the size and shape of a small walnut, and is lying at the top of the labium majus. It does not reach quite to the external abdominal ring, but its upper end points very close to it. You can move it from side to side, but you cannot pull it downwards. It evidently has some connection with the inguinal canal, but those of you who have told me that you think it is a hernia have apparently failed to notice that it has a definite upper limit, and that you can get the tip of your finger between it and the abdominal ring. If it were a hernia, you would not be able to do this, and, moreover, you would be able to make out a distinct stalk or pedicle passing from it into the inguinal canal.

If you take the tumour between the finger and thumb of each hand, you will be able to detect an obscure fluctuation. The fluid in the tumour is not pus; there is no sign of inflammation; the girl let you all examine without feeling any discomfort, and she says that the tumour has existed in the condition in which we now see it for very many years. Well, those of you who say that it is probably a collection of fluid in the funicular process of peritoneum which passed down with the round ligament of the uterus are probably correct; it is an encysted hydrocele of the cord, or, to speak with greater precision, an encysted hydrocele in the canal of Nuck. I propose

dissecting out the cyst next time we meet; dissection is the only satisfactory way of dealing with these inguinal cysts.

The tumour upon the other side is of far greater importance, and before proceeding to discuss its pathology we will get the dresser, to read his notes of the case.

From this report it appears that both parents of the young woman are alive and healthy, but that a sister died of "consumption." She has enjoyed fairly good health till the last four years or so; she has no recollection whatever of having met with an accident, and the worst trouble that she has had seems to have been some obscure neuralgic affection over the outer side of the left thigh and leg in the neighbourhood of the knee. For this she rubbed in "white oils," which are of much repute in stables, and consist, I believe, of a mixture of olive oil and of a strong solution of ammonia. The report has not given us a very lengthy clinical history, but it has probably told us everything that has any important bearing upon the case, and, at any rate, all that we need ask. But in describing her "present condition" there occurs a mistake of considerable importance, as I shall point out in due course. But first as regards the tumour; it forms, as you see, a prominent soft swelling at the base of Scarpa's triangle. It is not painful or tender, and the skin over it is unaltered in any way. One of you has suggested that it may be a sarcoma, and that the fluctuation which is very obvious in it is caused by the softness associated with extremely rapid cell growth, with hæmorrhage into or cystic degeneration of the sarcomatous mass. But this diagnosis is scarcely consistent with the history, for the girl says that she has noticed it coming on for a year or more. I commend this unusually cautious look-out for sarcoma, because sarcomatous tumours have a disagreeable way of discovering themselves when their presence is least expected, and they frequently offer a close and embarrassing resemblance to abscess. A surgeon who says that he has never been deceived in such circumstances has probably either had but little practical experience, or else he has a short or treacherous memory; there is another alternative. But this tumour is not a sarcoma; look, it is in communication with some obscure fulness in the iliac fossa, and, as the notes say, a distinct fluctuation can be made out under

Poupart's ligament, between the pelvic and the crural swellings.

Some of you have given me the diagnosis of "chronic abscess;" if by that you mean that the swelling contains *pus*, I cannot accept it as correct, for the word "*pus*" implies the presence of pyogenic micro-organisms, and there are certainly none such here. This is her temperature chart; it shows that there has been no disturbance whatever of her heat-producing centre. But if you employ the word "abscess" merely for convenience, and you mean that the material in the swelling is broken-down tuberculous granulation tissue, I gladly accept your view of the case. Then comes the question, what is the source of this tuberculous fluid? You do not seem inclined to accept the theory that it has travelled down from a carious patch at the front of the spinal column. Some of you have evidently examined the spine, but you do not seem inclined to regard this swelling as a *spinal* abscess. You apparently agree with the dresser's statement that the spine "moves freely in every direction." Well, if it did, this would not be a spinal abscess; but the dresser's statement is incorrect as I will shortly prove. Stiffness of the spine is the great, the early, and the persistent sign of Pott's disease. A tuberculous ulceration upon a vertebra, or upon two adjacent vertebræ, dreads disturbance almost as much as does a similar condition upon the articular surface of a femur or tibia, and the muscles in the neighbourhood are in a state of constant watchfulness against any movement being imparted to the affected vertebræ. If the disease happens to be in the cervical region, the complexus on each side of the spinous processes is rigid and prominent, and if the ulceration is in the lumbar region, the erector spinæ is usually in a similar state of apprehensive contraction. Each one of you ought to have inquired for stiffness in the girl's back, but most of those who did make that investigation apparently failed to detect any stiffness, probably because they do not know what is actually the amount of mobility allowed in a normal back. You have got no standard, as it were, by which to measure mobility. I will ask Sister, therefore, to pick out of her ward a girl with a healthy spine, which we may uncover and examine side by side with this girl. And while Sister has gone in search of such a patient, let me ask you if the reason of

so many of you missing the diagnosis in this case is because there is no angular projection in the girl's back? Remember the arrangement of the three curves in the spinal column; the most important is that in the dorsal region, with the convexity directed backwards, whilst the cervical and lumbar curves have their *concavity* directed backwards.

The result is that when the bodies of two dorsal vertebræ which have been weakened by an invasion of tuberculous granulation tissue fall together under the superimposed weight their spinous processes at once stand out conspicuously beneath the skin. But the effect of the falling together of the bodies of cervical or lumbar vertebræ is to efface the backward concavity of that region, so that a straightness of the spinal column in the cervical or lumbar region is just as pathognomonic of Pott's disease in the neck or loins as is the angular deformity in the case of dorsal caries. Now look at this girl's lumbar region; it is as straight as a line. In fact, from the pelvis to the lower interscapular region there is no trace of any curvature, nor will you be able to impart any to it.

Sister has now brought in a girl with a healthy spine that we may use as a standard. The girl is within a year of the age of our patient; she is in the hospital because she has run a needle into her foot. You shall see both backs in profile, and I will ask each girl as she sits on her couch to bend her head down towards her knees. See how smoothly and evenly the spine of the second girl bends, it shows a beautiful curve from the pelvis upward to the head in one continuous sweep, and she is able to put her head well down towards her knees, whilst her legs are flat on the couch. The difference between the ranges of movement of the two spines is obvious, for the vertebral column of our patient remains stiff and straight from the lower dorsal region to the base of the sacrum, flexion taking place in the upper dorsal and in the cervical regions only. By no effort can she get her head near her knees.

I will now ask one of you to lay his left hand over the lumbar spines and his right hand over the cervico-dorsal region of each patient in turn whilst I ask her to bend and straighten her back; he will tell you that in the lumbar region of the affected spine there is no movement whatever—that the loins are as stiff as a board. Now I will place the

patients with their backs towards you, and will ask you to notice the difference in the movements when, with the pelvis fixed, I ask each girl to incline her trunk sideways to the right and to the left. The affected spine shows no movement in the lumbar region; but look how freely the other inclines to either side.

A very excellent test for the amount of mobility in the spine is to fix the pelvis and then to get the patient to turn and look over her right or her left shoulder. See how awkward at the exercise is the girl with tuberculous disease, whilst the other is able to rotate every vertebra upon its fellows, and with perfect ease and natural grace. This is, in my opinion, the most delicate test for movement in the vertebral column that one can employ when searching for disease.

Lastly, we come to the question of pain. Were some of you afraid to diagnose Pott's disease because the girl told you that never at any time had she noticed pain in her back? The belief that pain is always associated with spinal caries is a very common error, and in order to free your mind now and for ever from such misapprehension, I will ask the patient before you once again if she has ever had pain in the back, back-ache, lumbago, or anything of the kind. To each question she gives positive assurance in the negative sense. She insists that the only pain which she has ever had was the "rheumatic" trouble in connection with the terminal filaments of the external cutaneous nerve. In many cases of Pott's disease, though the complete absence of local pain is noted, a history of peripheral pains may be elicited. The great feature of these peripheral pains is that they are nearly always symmetrically distributed in the two sides of the body, and that for the most part they concern the remotest peripheral filaments of the nerves implicated. Thus, to be brief, in dorsal caries the pains are referred by the terminal filaments of the intercostal nerves to the front of the chest or to the skin in the epigastric and umbilical regions, and in lumbar caries they are referred to each hypogastric, inguinal, or crural region; to the area of distribution of the ultimate fibres of external, middle, or internal cutaneous nerve; or over the ball of each great toe, where the internal saphenous nerve ends. I say again the pains are referred with characteristic evenness to the two sides, and *symmetry in the area of pain should always make*

one look for a central source of trouble. The pains are referred evenly to the two sides of the body for the simple reason that tuberculous ulceration begins at the very front of the vertebra, and spreading laterally, involves the dura mater or the posterior roots of the spinal nerves evenly in inflammatory pressure. But, as you have noticed, no amount of cross-examination has enabled us to obtain from this girl the admission that she has had symmetrical pains. No; she insists that she has never had any pain whatever except the "rheumatism" on the outer side of the left knee.

If you have been following me closely you ought now to be feeling a little confused; you ought to be saying to yourselves, "Our friend has been insisting that Pott's disease may exist without any complaints of pain in the back, and he goes on to say that the pains due to inflammatory pressure near the carious region are experienced evenly upon the two sides of the body. Here is a case of Pott's disease in a girl who has certainly had no pain in the back, though she has had peripheral pains in the region of the left external cutaneous nerve, but, for certain, only on one side; there must be a fallacy." The explanation is this: she has had no pains in the back, and those "rheumatic" pains were not due to inflammatory pressure upon the nerves in the carious region (if they had been they would for certain have been symmetrically distributed); they were due to direct pressure upon the external cutaneous nerve by the large "abscess" in the iliac region. When one sees how full the iliac fossa is, it becomes almost a matter for surprise that more nerves were not disturbed by the increasing accumulation. But we must accept clinical facts as we find them.

Though I call this collection of fluid an "abscess," I have clearly explained, I hope, that it is not an abscess. It is a broken-down fluid mass of tuberculous granulation tissue. But it is inconvenient to call it by so long a name, and the term "abscess" which was given it by our predecessors has the merit of being short and handy, and that is its only merit. It is no more an "abscess" than is the broken-down, soft, and fluctuating node which may be found upon the forehead of a syphilitic woman. And I am sure that you would not like to call a broken-down syphilitic granuloma an "abscess."

Then as to the anatomy: the "abscess" is due

to a mass of granulation tissue, finding its way from the carious patch under the anterior common ligament outwards beneath the fascia which binds down the psoas. The granulation tissue slowly takes the place of the psoas and gradually breaks down into a curdy fluid. The fascia which binds down the psoas covers in the iliacus as well, so it is small wonder if the "pus" treats the fibres of the iliacus as it treated those of the psoas. An iliac "abscess" bulges above Poupart's ligament near to the anterior part of the iliac crest, as you see this bulging; a psoas "abscess" causes a fulness near the spine, and then finds its way behind the crural sheath into the thigh, as this has done. This collection is therefore a psoas, an iliac, and a crural "abscess." Moreover, if you feel between the last rib and the back of the iliac crest you will find that it is trying to become also a lumbar "abscess."

Having insisted so much upon the exact median arrangement of the carious patch, you may expect me to try to explain how it is that the granulation tissue in this case has crept out beneath the left side of the strong anterior common ligament, and not at the same time beneath the right side. We must accept clinical facts as we find them. It must be, I suppose, a matter of anatomical chance; not infrequently the granuloma emerges from beneath both lateral borders of the ligament, a psoas "abscess" being formed on each side of the body. Two such abscesses communicate with each other across the carious patch beneath the common ligament, and when they are opened an antiseptic fluid injected into one will escape by the other. And if a person has double psoas abscess, each side should be dealt with surgically at the same time, for the two abscesses are actually but lateral offshoots from the one central abscess or ulceration.

In the active treatment of this large spinal abscess the greatest possible care must be taken—the area of operation, the surgeon's fingers and his instruments must not only be cleansed but *clean*. To introduce, or to allow of the introduction of any septic germs into the cavity would be to invite dire disaster. Some of us can remember the time when pretty nearly every case of spinal abscess went wrong, the patient dying of septicæmia or pyæmia.

Whether the surgeon opened the "abscess" or

left it to nature to effect an opening by ulceration, the result was the same—profuse suppuration, exhaustion, hectic, and death. Now happily it is entirely different, although from time to time abscesses which have for awhile been discharging a thin fluid are unfortunately allowed, by the supineness of the surgeon, dresser, or nurse, to become inoculated. It must always be remembered that a leaking aseptic spinal abscess needs careful and untiring aseptic treatment every bit as much as does one which is just being opened.

The fluid in this cavity is walled in by a layer of tuberculous granulation tissue which, in various parts, springs from bone, ligament, muscle, and fascia, and the treatment consists in getting rid of the fluid and in scraping away the unhealthy granulation layer—the pyogenic membrane, as it used to be called,—washing out the cavity with warm lotion which may have a destroying effect upon the lurking bacilli tuberculosis, swabbing out and drying the cavity, and then sewing up the wound.

Someone has just asked if after opening this collection I propose leaving in a drainage-tube. To that question I reply “Certainly not.” If this were a septic abscess I would probably leave in a tube; but it is not septic, and not an “abscess,” so, having cleared it out, I shall sew up the wound tight. Since the surgeon has understood that these “cold abscesses” are merely broken-down tuberculous granulomata, their treatment has greatly improved. Treated on modern principles they may be expected to heal up straight away, and they obviously could not do this if a tube were left in them.

As regards the lotion for irrigation, I know of nothing better than a hot solution of zinc chloride, of the strength of ten grains to the ounce. And if, as is probably the case, there are bacilli lying deep in crevices of carious bone, or between strong bundles of ligament, so that they cannot be reached by the scraping or be shrivelled up by the lotion, is the radical treatment of the “abscess” doomed to end in disappointment? By no means. Yet a short while ago it used to be taught as pathological gospel that such would be the case. If any of you to-day hold to such a worn-out belief, let me ask you, if tuberculous disease which had never had active surgical treatment ever got well of itself? If logical, you ought to say “no.”

Then, what about those shortened people with a prominent hump between their shoulders, whom you meet in the street. In their childhood or youth they were the subjects of Pott's disease (which is undoubtedly tuberculous), and they have entirely recovered from it (except for the deformity); and having fortunately escaped without abscess they have had neither irrigation nor wound. And those children with lingering synovitis of the knee (undoubtedly tuberculous) which, without the aid of arthrectomy or injections, have triumphed over their disease and regained a useful joint—do not they also show the fallacy of the theory that unless all the bacilli have been individually and actively attacked treatment is in vain?

It seems to me that we have occupied ourselves so much with theories, and have formed so exalted an opinion of the value of active interference in cases of tuberculosis, that we have almost forgotten the existence of the *vis medicatrix nature*, but happily she still remains with us!

In this case we have to deal with a cavity which extends from the top of the psoas to the middle of Scarpa's triangle. How shall we open it for the scraping? One of you has said, “Make a single incision in the most dependent part, a little below Poupart's ligament.” In that case the top of the cavity will be very far distant from the opening, and to scrape it out we should need a spoon as long as that to which Mr. Chamberlain suggestively referred the other day. I think it may be well to make one incision there through which we can scrape out the lower part of the cavity, and, having done that, we can sew it up tight. But it will be necessary to make a second incision in the interval between the last rib and the back of the iliac crest, working our way into the cavity by the guidance of one of the lumbar transverse processes. Having done this, and having introduced our finger, we should find no trace of psoas, but we should very likely be able to make out the cords of the lumbar plexus stretching like harp strings through the tuberculous fluid, and covered by a layer of granulation tissue, for though muscular fibres quickly disappear under the disintegrating force of the granulation tissue, the nerves are possessed of a much greater power of resistance.

The scraping will have to be done very tho-

roughly, but not roughly, and the irrigation will have to be carried on until the fluid comes back quite clear. Then, after the wound is sewn up, and dry dressings are applied under a binder, we shall hope for a complete, though possibly a tardy recovery. Before the girl leaves the hospital she will be fitted with a stiff jacket of some sort so as to keep the diseased vertebræ at rest, for *rest* is the one and the only line of treatment for the tuberculous ostitis.

For what Period of Time can Immunity from Diphtheria be Conferred by a Single Injection of Antitoxin? The Dosage.—F. G. Morrill ('Boston Medical and Surgical Journal,' March 3rd, 1898) arrives at the following conclusions:

1. That immunity in any given case, of no matter how thorough exposure to diphtheria, may be conferred for at least ten days by the injection of a small dose (100 to 250 units) of serum, provided it is given twenty-four hours previous to actual infection.

2. That a larger dose (250 units for a child of two up to 500 units for one of eight or over) will confer safety for three weeks—or, to be a little more conservative, let us say twenty days—under similar conditions.

3. That no harm will result from the treatment in a vast majority of cases of sick children, probably in no case of a healthy child, provided the serum used is up to the present standard of purity.

In conclusion, he says that any one who thinks that antitoxin will prevent the occurrence of a follicular tonsillitis or of a coryza in an individual who happens to have the Klebs-Löffler bacillus in his throat or nose will be disappointed, for neither of these conditions constitutes a diphtheria, any more than the co-existence of the pneumococcus in the saliva, and a bronchitis constitutes a frank pneumonia. He adds that a physician who fails to promptly immunise the members of a family or close community in which diphtheria breaks out neglects to do his duty by those whose safety lies in his hands.

Charlotte Medical Journal, June, 1898.

DEMONSTRATION OF CASES AT THE NORTH-WEST LONDON CLINICAL SOCIETY,

Dr. ALEXANDER MORISON in the Chair.

MR. JACKSON CLARKE showed a young man, æt. 30, who had been suffering from the sequelæ of gonorrhœa for thirteen years. He was sent in for extravasation of urine, and when Mr. Clarke first saw him his scrotum was the size of a human head, and he was suffering great pain. In the perinæum was a scar from a previous operation, no doubt resulting from the opening of a small peri-urethral abscess. The urethra was found to be densely and narrowly constricted, and there was acquired phimosis. The constriction was so great that he could not get in a grooved staff to guide him in the operation. He therefore had to proceed as follows: he introduced the finger into the rectum and felt the apex of the prostate and did a Cock's operation. The patient had so completely emptied his bladder into the tissues that no urine came out through the incision. He then extended the incision forwards as far as the beginning of the scrotum, and it was incised in the brawny tissue to a depth of three inches. That revealed the exact state of affairs. There was a large peri-urethral abscess so that the whole of the spongy part of the urethra lay as if dissected out in the abscess cavity. He next dealt with the urethra; that was so tightly constricted that even a fine catheter would not go beyond three inches. He passed a little catheter for that distance, cut down on the end of it, and then did Wheelhouse's operation; that is, opened the constricted urethra another three inches. The communication between the abscess and urethra must have been a very small one, for it was not visible in that proceeding. He noticed some small calculi free in the abscess cavity, and embedded in the inflammatory tissue of the altered urethra were calcareous nodules. No doubt the first abscess had been caused by the perforation of one of these small urethral calculi. The operation was nearly at an end, but the whole scrotum was so œdematous that two more large and deep incisions were made, one on each side of the middle line in

the scrotum. Then came the question of how to deal with the constricted anterior part of the urethra, the penile portion was left to gradual dilatation with the catheter after the operation. After washing the large cavity out well and removing all the granulation tissue, the patient was put to bed and the wound packed with iodoform gauze. Mr. Jackson Clarke did not at that time tie in a catheter. The walls of the urethra were so well preserved that he left the future reconstruction of the urethra to catheterisation later on. He did not like to tie in a catheter where he could avoid it. The operation was done on the 6th February. For two days the patient had a sharp rise of temperature, which was accounted for by the sloughing of some of the tissue on one side of the perinæal incision. When that slough separated the temperature became normal, and remained so nearly all through. Catheterisation was begun the second day after the operation, and regularly continued up to the present time; subsequently circumcision for the phimosis, myotomy, and then gradual dilatation with catheters daily, until a No. 14 could be passed. The progress of the case was good, except that on one occasion the temperature rose owing to the re-formation of an abscess when the wound was nearly closed, but by injecting weak carbolic acid down the urethra the suppuration was got rid of. He had now no suppuration, and the wound in the perinæum was healed. He now passed a No. 13 catheter for himself easily.

Mr. KEETLEY said he had had the misfortune, the other day, to lose a patient who had extravasation of urine and consequent abscess. The patient, a man of sixty, was in a "typhoid state" when he (Mr. Keetley) was consulted. The extravasation extended along the upper side of Poupart's ligament into the flanks. He noticed that young medical men, who had not seen many of those cases, were apt not to look for that; it was not uncommon, and was a very serious thing if overlooked. The subcutaneous cellular tissue, in the left flank especially, was one large black slough, and although free incisions were made, and both the urethral and perinæal aspects of the case radically treated, the patient died. But patients did not always die, even when they had disease as extensive as this. Some years ago there was a policeman in the hospital in whom the extrava-

tion extended up to the axilla, and on the right side it was necessary to make an incision which extended from the pubes to the apex of the right axilla. He (Mr. Keetley) showed the case at the Medical Society some time afterwards. The patient recovered, and for many years did his duty as a policeman. The speaker did not think any harm was done by fixing a catheter passed from the perinæal wound to the bladder. There was only about the length of a finger from the surface of the perinæum to the bladder, and there was plenty of room for a good large india-rubber catheter to lie quite easily, which was especially useful if the bladder had to be washed out frequently.

Dr. MACLURE showed a woman, 24 years of age, the subject of insufficiency of pulmonary and other cardiac valves. The case came under his care one and a half years previously with the ordinary symptoms of cardiac trouble—anasarca and attacks of pain at night over the heart, shooting down the left arm, with insomnia and uncontrollable vomiting. She also had complete retention of urine for nine months. Catheter was passed by patient herself once in three days during this time. Gradually she got weaker and wasted very much, so that she had to remain in bed. That went on for nine months, and last March she had an attack of acute mania, which lasted about a fortnight. At the end of that time she seemed to get better, and in the course of a week or two she gradually got up, and was now able to go about. She could not walk very far without suffering from shortness of breath, and if she walked more than half a mile she again had anasarca, but twenty-four to thirty-six hours' rest caused it to rapidly disappear. She had recovered power over her bladder, and the catheter had not been used for three weeks. She had suffered from cardiac symptoms as long as she could remember, and had had three attacks of rheumatic fever, the first when she was sixteen years of age. At seven years of age she had whooping-cough, scarlet fever, and diphtheria.

Present condition.—She has an ill-defined fullness over the left supra-clavicular region and under the trapezius muscle. A to-and-fro murmur can be heard, loudest in the pulmonary area, and conducted downwards to the left of the sternum. Apex-beat in fifth intercostal space in nipple line.

Presystolic thrill at apex, with other evidence of double mitral disease. The absence of Corrigan's pulse, capillary pulsation, and of much hypertrophy and dilatation of left ventricle, is in favour of pulmonary rather than of aortic disease. Since the mania in March she has had loss of sensation and loss of power in the ring and little fingers of the left hand, and the loss of sensation is tending to extend upwards over the back of the hand. Her left pupil is very much larger than her right. She still suffers very much from insomnia and pain.

Dr. HARRY CAMPBELL said he heard a diastolic murmur loudest to the left of the sternum; in the second or third interspace the murmur could be traced down to the apex. There was no marked pulsation of the carotids and no typical "aortic" pulse, nor any decided hypertrophy and dilatation of the left ventricle. This rendered it somewhat doubtful whether it was an aortic murmur. He did not hear a presystolic murmur, but as there was a thrill, and as the first sound was short, he thought there was mitral obstruction. He thought it was a case of slight aortic regurgitation *plus* mitral obstruction. He had never seen a case of pulmonary regurgitation.

Dr. G. A. SUTHERLAND differed from Dr. Campbell in his conclusions. He thought there was a presystolic thrill and a definite presystolic murmur of considerable loudness, distributed over a wide area. He traced it to the pulmonary area, and was inclined to regard the murmur audible there as the presystolic murmur of the apex conducted upwards. He wished to ask Dr. Maclure whether, assuming that there was a presystolic murmur, that would sufficiently explain the symptoms from which the patient suffered. As regards the exocardial symptoms, the condition of the pupil, the swelling in the neck, and the symptoms in the arm, those must be referred to a different pathological lesion, probably some tumour in the supra-clavicular region with pressure upon the nerves.

Dr. L. A. PARRY said the impression he got from examination of the patient was that it was a case of mitral obstruction. He would have called the murmur late diastolic. He suggested as an explanation of Flint's murmur, referred to by Dr. Campbell (the presystolic murmur heard in the mitral area in some cases of aortic stenosis, when the necropsy showed no abnormality of the mitral

valve), that there was functional mitral obstruction, due to the increased intraventricular pressure which is present in aortic stenosis, and thus increased difficulty for the blood to pass from the auricle to the ventricle through the auriculo-ventricular orifice.

Dr. MORISON thought there was not so much insufficiency of the pulmonary valves as that the case represented the condition to which Dr. Sutherland had referred, namely, mitral obstruction. Pulmonary regurgitation, as Dr. Campbell had said, was a very rare condition. He had met with a case in an adult many years ago, and at that time he looked up the literature of the subject, but could only collect about twelve non-congenital cases. His own case was that of a man of twenty, who had had scarlet fever when he was young, and there was a marked to-and-fro murmur in the pulmonary area. The patient ultimately died, and he showed the specimen to the Pathological Society, a committee of which was appointed to examine and report upon it. One of the marked features of the bruit, in addition to its roughness, was its extreme localisation and superficiality; in fact, he might almost say such cases could be diagnosed by palpation alone. To account for unilateral dilatation of the pupil in the absence of pressure was extremely difficult. It must be borne in mind that the girl had been extremely neurotic, and had had an attack of mania, and that the condition was possibly what, for want of a more precise term, he conventionally called hysterical. The general diagnosis he would make, as far as his knowledge went, was that the case was one of mitral constrictive disease, the result of rheumatic fever.

In reply, Dr. MACLURE stated that the patient earned her living by fine needlework, and that the dilatation of one or both pupils was very embarrassing to her. He also considered that the point of maximum intensity and ring of the murmurs at left base of heart pointed to disease of pulmonary orifice rather than to mitral disease pure and simple.

Mr. KEETLEY showed a girl, seventeen years of age, who had been operated upon five years before for tubercular teno-synovitis of the sheath of the tendo Achilles. That sheath was full of pus, and there was a cavity extending up almost as high as the popliteal space, and it was lined by a thick pyogenic membrane. He believed the locality was a rare one to be attacked by the disease. She

seemed to have remained well during the greater part of the five years since the operation was performed, but recently she had complained of pain just outside or below the outer ankle; but the pain was in the position in which that of flat-foot was usually situated, and he was inclined to think it was due to flat-foot, which she undoubtedly had. He did not believe it was a recurrence of the tubercular disease. The ankle-joint itself seemed healthy. She was a servant girl, and therefore had much standing to do.

Mr. TEMPLETON thought there was no return of the disease in the tendo Achilles sheath, but he thought there was some disease in the sheaths of the peronei muscles. It was quite exceptional for the disease to persist as a tubercular teno-synovitis pure and simple; mischief generally extended into the neighbouring joints, especially in the case of the wrist. He drew an analogy between the state of things in the present patient and that in psoas abscess. He advocated rest and fixation in a splint as being more suitable than counter-irritation in cases of tubercular teno-synovitis.

Mr. KEETLEY showed a man who had sustained a comminuted fracture of the left patella two months before by slipping off the curb. He (Mr. Keetley) sutured the patella seven weeks ago. He perforated the two pieces subcutaneously, which obviated the necessity of handling the fragments so much, owing to their being covered up by the skin. But before inserting the wire he freely opened the joint, both to clear out the clots and to adjust the fragments of the comminuted bone. He exhibited the instruments with which he wired the patella. The lower fragment of the patella was broken into three pieces. Any form of purely subcutaneous suture in that case would have jammed three irregular fragments against the upper fragment. The man now had a very good joint.

Dr. SUTHERLAND showed a boy, aged 9, the subject of heart disease. The patient had been under observation on and off for the past fifteen months. There was no definite history of rheumatic fever, but there was a history of pains in the joints and in the precordia, and short attacks of ill-health characterised by shortness of breath and weakness. There was nothing special in the family history. A peculiar bright redness was to be noticed about the cheeks, nose, and chin, and a slight dusky blueness about the lips and hands,

together with some clubbing of the fingers. There was also to be noticed a very marked bulging in the front of his chest, which extended right across the middle line, being greater on the left than on the right side. Pulsation was also visible over a very large area, the pulsation corresponding with the impaired resonance over the precordia. This area extended from the right nipple to the left mid-axillary region, and measured eight inches. At the apex of the heart a distinct thrill could be perceived, systolic in time, and the beat was most forcible outside the nipple in the fifth left inter-space. At that point he could hear a double murmur, one part of which was systolic and the other part diastolic or presystolic. Towards the base of the heart, over the pulmonary area and extending across the sternum and occupying the whole of the position of dulness on the right side, there was a definite to-and-fro rub, apparently of exocardial origin. Sometimes a definite cantering rhythm over the heart could be heard. The pulse was regular and somewhat small. As regards the associated conditions the boy did not appear to suffer very definitely from cardiac symptoms, and there was not more than the very minutest trace of œdema in the extremities. There had never been any albuminuria or ascites. The liver was somewhat enlarged. The boy had a little cough at times, and a slight pulmonary catarrh. He could run about fairly well, but much exertion made him short of breath. As regards the exact nature of the cardiac affection the complexion suggested the possibility of the mischief being congenital; he thought the colour of the boy's face was more like that which was seen in the subjects of mixed blood through congenital defects. He had no doubt the boy suffered from acquired endocarditis and pericarditis, but he thought the muscle of the heart had not suffered so markedly as might be expected in a case of the sort; there was an unusual absence of symptoms of myocarditis. A possibility was that there was a chronic inflammatory process going on, and that the boy was suffering from a condition of mediastino-pericarditis which was extending in all directions, involving the surrounding structures and matting them together.

Dr. McCANN showed pathological specimens illustrating extra-uterine gestation. The first patient was a lady aged 27, who had been married

three years, and was sterile. She had suffered all her married life from intense dysmenorrhœa, being confined to bed the first day of every period. She consulted him on November 16th last year, when she told him that three weeks previously she had had a sudden attack of pain in the left iliac region, with sickness and faintness, at which time she had missed her period for a fortnight. Shortly after marriage she had had an attack which was diagnosed as inflammation of the bowels, which confined her to bed for a fortnight. Before he saw her she was attended by a local doctor, who thought she had another attack of inflammation of the bowels, and she was kept in bed for ten days, when she was able to come back to town. This patient's husband had a chronic gleet when he was married, and the fact of this so-called inflammation of the bowels coming on shortly after marriage made him (Dr. McCann) suspicious that she had had an attack of gonorrhœal salpingitis. The patient was getting gradually thinner. On examination he found to the left of the uterus an elongated swelling, tender and fluctuating; to the right of the uterus a prolapsed ovary, with considerable tenderness on that side. His opinion was that something had been leaking into the abdomen, and from the facts of the case he believed it was pus from a pyosalpinx. He operated upon her on November 20th, and found a swelling on the left side which was adherent, and in getting it up through the wound it ruptured, and a portion of the contained clot came away, which was afterwards found and examined. It would be seen from the specimen that the abdominal end of the tube was unusually patent; there was an egg-shaped swelling, which contained an egg-shaped clot; in the centre of the clot was a cavity. After a most careful examination of the tubal wall and the clot he had not been able to find any trace of chorionic villi, yet he believed it to be a very early tubal gestation. There was no passage of decidual membrane.

The second case was more advanced. The patient was aged 37, and had had four children, the last two years ago. In November last year she had continuous pain in the right iliac region, and pain at the umbilicus. There was a blood-stained vaginal discharge. The temperature varied from 98° to 101°. On the 6th December, when he saw the patient, her temperature was 101°; she was very blanched, and complained of

intense pain in the right iliac region, with vomiting. The abdomen was distended. A well-marked sausage-shaped swelling was found on the right of the uterus, the uterus being directed to the left, forward, and enlarged. In addition there was what felt like another fluctuating tumour, but it was really blood. On opening the abdomen he found a swelling on the right side of the uterus, and a large mass of blood-clot at the fimbriated extremity of the Fallopian tube. The tube was somewhat adherent. He clamped the broad ligament and ligatured and removed the tube. On examination of the appendages on the left side he found the tube thickened. He separated the adherent left tube. The abdomen was flushed with saline solution, and the abdominal wall stitched. The patient made an excellent recovery. At one time it was supposed that the patient had appendicitis, and that the swelling on the right side was really an abscess connected with the appendix, but on examining under anæsthesia it was fairly clear that it was a tubal swelling. She had missed her menstrual periods for three months. There were present several of the signs usually associated with tubal pregnancy, viz. cessation of menstruation, enlarged breasts, sudden acute abdominal pain associated with sickness, and pallor.

He operated on a patient three weeks before who gave him the following history. She had missed her periods for two months, she said her breasts were enlarging, she had had one or two attacks of pain and sickness, the pain being in the right iliac region, and she fancied she was pregnant. On examination he found a rather movable swelling about the size of a lemon to the right of the uterus, and in addition most marked pulsation on that side. He thought it possible that this might be another case of tubal gestation. On opening the abdomen he found it was not a tubal foetation, but a dermoid cyst with a twisted pedicle. The history and physical signs almost exactly resembled those of tubal pregnancy.

Dr. Gow thought the cases brought forward by Dr. McCann showed very well how difficult it often was to distinguish between extra-uterine gestations and pelvic inflammations, and he was not surprised that there seemed considerable doubt in Dr. McCann's mind as to whether he was dealing with an inflamed tube or an extra-uterine gesta-

tion; it was a mistake which constantly arose, and could sometimes hardly be avoided. Out of seventeen or eighteen cases which he had operated on, the majority of them were cases of tubal mole. Sometimes cases were met with where rupture had taken place into the abdominal cavity, but they were comparatively rare. In the vast majority of cases of tubal gestation the ovum died very early and became converted into a mole. He thought the first case was really one of salpingitis with hæmorrhage into the interior of the tube, whilst the second specimen was a good example of a tubal mole. Dr. McCann had spoken about "sausage-shaped swellings," but he believed that, as a matter of fact, most of the tubal swellings felt spherical, for the simple reason that the tube was coiled round the ovary. Of course the swelling might be sausage-shaped, but he thought a sausage-shaped swelling, as the result of a distended tube, was unusual.

Dr. JOHN SHAW-MACKENZIE said an interesting point was the frequent impossibility of diagnosing tubal rupture before operation.

It had been said that no abdominal section should be done for purposes of diagnosis, but he was sure there were cases which proved to be ruptured tubal gestation, and in which it was impossible to be quite sure of the condition before operation. As Dr. McCann pointed out, such cases often simulated leakage of pus from the tube more than anything else.

Last August a case was admitted into the North-West London Hospital, presumably of suppurative peritonitis, under Mr. Jackson Clarke. He (Dr. Shaw-Mackenzie) in the holiday absence of Dr. John Shaw, considered the usual symptoms of ruptured tube, and thought he had negatived such condition. In any case immediate section was indicated, and Mr. Jackson Clarke opened the abdomen. It proved to be a case of septic peritonitis (puerperal fever) three days after rupture of a gravid tube. In this case the classical signs of ectopic gestation, viz. previous sterility and bimanual pelvic tumour, were absent. Moreover, there was unusually high temperature, and no history could be obtained from the patient, and none had been sent with her. In going over the histories subsequently of forty-five cases reported in various places, he found that in seven of these previous conception had occurred within the year,

so that previous sterility was not always a reliable sign. The absence of bimanual tumour was explained by the amount of free blood in the peritoneal cavity. He thought pulsating vessels over a bimanual tumour sometimes diagnostic previous to rupture, but they might be present under other conditions or absent in cases of collapse after rupture. He agreed with Dr. McCann in the presence of previous peritonitis in many of these cases. He inclined to a constitutional origin of salpingitis in such cases, and he would like to ask Dr. McCann if the gleet mentioned in the husband of one of his cases might not have been a syphilitic urethritis rather than gonorrhœa? Upon such supposition he thought the tendency to septic changes in the effused blood great, and a drainage tube after operation for tubal rupture advisable.

NOTES, &c.

TECHNIQUE OF A NEW HYDROTHERAPEUTIC METHOD.

By FENTON B. TURCK, M.D.

IT is often a perplexing question with practitioners how to devise an effectual method for inducing peripheral circulation and the reduction of congestion of the viscera in cases where all ordinary methods fail. More especially is this necessary where the resources at command are limited, or where it is impractical for patients to visit institutions devoted to hydrotherapy. I find that textbooks deal with hydrotherapy in the most general terms, referring to warm baths, cold baths, fan douches, rain douches, with but meagre technical descriptions. If one may judge, the writers have had little or no practical experience. In this paper the writer aims to direct practitioners to a simple, novel, and effectual method of attaining the desired end with the ordinary means at command.

Hydrotherapeutics, as practised in many sanitariums under the direction of unskilled attendants, without the personal direction of skilled physicians, is promotive of much harm and disastrous results. Under proper direction it is productive of great and lasting benefit; but it must be practised with

a rigorous attention to technique and under proper limitations. To boldly assert, as does one writer, that "the chief aim of hydrotherapy is to stimulate and give tone to the nerves from which all vital energy emanates," may be admitted as a generalisation, but how and when to apply it requires a thorough knowledge of the subject.

In the 'Wiener med. Woch.,' Nos. 1 and 2, 1895, I presented a method of hydrotherapy which has for its principal object the reduction of the congestion of the viscera, especially of the splanchnic area, and a more equal distribution of the blood over the body. As this congestion is a frequent complication in many cases of chronic gastro-enteritis and other diseases, a prompt and effectual method is essential in our therapeutic armament.

Among the many functions of the skin as an organ is the detection of different degrees of temperature in contact with the surface. The different temperatures principally manifest themselves by increasing or diminishing the temperature of the skin, and thus in some way stimulate the end nerves and filaments of certain sensory nerves which may be termed temperature nerves. The skin varies in degree of sensitiveness in different parts of the body. The face is more acute than the hand, while the trunk and the limbs are least sensitive. As in other organs of special sense, the end filaments of temperature nerves seem adapted only to a certain form of stimulus. When a stimulus passes beyond that for which the temperature sense is adapted, it ceases to transmit the sensation; the temperature sensation disappears, and raising the temperature gradually the heat is not felt, as the skin, to use a common phrase, becomes used to it. When the temperature has reached 115° or 120° F., the skin still does not convey the sensation of heat, but may even carry a sensation of cold, and a feeling, as a chill, may for a moment pass over the body, the skin showing oftentimes the appearance of "goose flesh," at times noticed along the outer side of the thighs. If the patient is left in the bath too long to cause an accumulation of heat, he may suffer from heat-stroke, but not so much from the irritation or burning sensation of the skin, as the end nerves now seem partially paralysed, not only to the sense of heat, but to the sense of touch, pain, and other common sensations of the skin. At

this period of the bath examination of the abdominal organs is easily conducted, due to the relaxation and anæsthesia of the abdominal wall. Ice may then be applied to the surface and cause no shock or sensation of cold; yet the blood-vessels receive the stimulating and beneficial influence.

The exact technique of the following described bath I have not found in literature. The essential part of the hot-water bath, followed by ice massage, is the technique. There is nothing new in the use of water at high temperature, or in the use of ice. The patient is submerged up to the neck in a bath at a temperature of 100° F.; a short piece of hose is attached to a hot-water faucet; the hot water is turned on, and the hose is swept from side to side, up and down through the water, to rapidly diffuse the hot water throughout. As the temperature of the bath rapidly rises to 110° F. the patient is instructed to lie perfectly quiet. Movements like reaching the arms out of water will give a burning sensation, while if the patient remains quiet the contrast in temperature is not so markedly realised. As the temperature rises to 112° , 114° , and 115° F., the pulse and respiration are observed. If at this temperature the whole surface of the body becomes a crimson red, the patient may be removed. The time required averages about ten minutes, and the temperature about 114° F. If after ten minutes' time has elapsed the temperature has reached 115° F., the surface of the body does not yet show evidence of injection of the peripheral arterioles, "flushing of capillaries," the temperature may be increased if the condition of the patient permits. Water at high temperature is a stimulant, while at lower temperatures it may become a depressant. I have in some cases given a bath up to a temperature of 125° F. It is essential that the surfaces of the body show arterial injection to gain the full value of the treatment to follow. The patient is then removed from the bath and made to sit on a board placed across the bath-tub, the feet extending and resting at the edge of the tub. A piece of ice, weighing a pound or two, is rubbed over the head, face, and neck, then massage over the back up and down, over the breast and abdomen, and arms and legs. This is repeated rapidly, and the patient soon shows signs of exhilaration and a general good feeling.

If these instructions are properly carried out

there is no shock from the use of the ice, as stated. Sometimes I give an ice-water douche under pressure if there are no contra-indications, but this even is not beneficial in a large majority of cases. In many instances a douche, either hot or cold, or alternated, is better left out, for the bath such as I have described will meet the indications. If a douche is given, and there is no mechanism in the plumbing for regulating pressure, the very simple device of a garden pump is used, which can be purchased for about six dollars. If exactness under pressure is desired, a compressed air tank is used, the closed irrigator filled with water and connected with a compressed air tank, so that the air will force out the water through the hose connection, and thus produce a forced douche. A manometer connected with the irrigator will give the pressure that is being projected. Another method I have before described is the use of a large horse syringe for projecting ice-water.

The effect of this bath is to stimulate the skin, and reflexly the vaso-motor system, whereby the blood is withdrawn from the congested viscera and distributed over the surface of the body. The time required for producing a "glow" upon the surface of the body varies in different individuals, depending upon the condition of the circulatory system, as well as the rapidity and increase in temperature of the water. One thing is certain, almost every physician can carry out this simple hot-water bath, followed by ice massage, as described, and he can study his cases better. He can have it used upon himself when tired out and depressed by overwork and errors of living, and he will then be able to appreciate and apply a simple and effectual measure in the treatment of a selected class of cases.

As to contra-indications, the bath is to be avoided when there is marked fatty heart, diseases of the arteries, or any tendency to apoplexy; but in many cases, when either warm or cold baths given singly are contra-indicated, the bath I have above described can be used with great benefit.—*Louisville Journal of Surgery and Medicine*, July, 1898.

COHN considers that the heat of summer may disturb the digestion of infants in some unknown way, and this must be considered as a factor in the ætiology of diarrhoea in artificially fed children.

Arch. für Kinderheilk.

The Use of Manganese in the Treatment of Dysmenorrhœa.—Dr. O'Donovan, writing of his experiences with manganese in dysmenorrhœa, tells us that he published an article entitled "A Plea for the Use of the Manganese Compounds in Certain Forms of Dysmenorrhœa," in which he drew the following conclusions:

1. The manganese compounds are valuable additions to the therapeutics of dysmenorrhœa, and in a certain number of properly selected cases great benefit may be expected from their use.

2. Their use does not interfere in any manner whatever with the administration of iron or the vegetable tonics, but rather aids and is aided by them.

3. The best results from the employment of these remedies may not be obtained at once, and failure should not be confessed until after a continuous trial lasting three months.

4. So far as we know at present, the black oxide of manganese is the most convenient form for administration.

At that time the observations of Ringer and Murrell, of London, called attention to the benefit to be derived from the compounds of manganese in various menstrual disorders, and other clinicians had testified in their favour. Having enjoyed a certain vogue at the time, in the avalanche of new remedies which are being constantly exploited manganese seems to have lost its prominence, and is now seldom referred to. This is a mistake—a remedy, valuable in a number of cases, is being overlooked. That it is one which deserves to be rescued from oblivion and again presented for study and trial is undoubted. The writer has been using manganese constantly since the appearance of the paper quoted, and is to-day as favourably impressed with it as when he reported his former observations. He holds it to be most valuable in the treatment of dysmenorrhœa in unmarried women; and, until it has been faithfully tried during a period of not less than three months without intermission, he would use no other treatment.

Manganese will not relieve all cases; in some it produces no effect whatever, and in some few instances women have complained that their sufferings actually seemed to have been exaggerated; but when it does act favourably, it is such a boon that it cannot be over-valued. Unfortunately, one

cannot foretell in a given case whether or not manganese will be of benefit; the writer has known it to fail when he had most reason to expect success, and to relieve when he feared it would fail, so that its use is somewhat empiric, and apt to lead to disappointment. This does not, however, change the fact that a large number of cases of dysmenorrhœa may be relieved by its use in a way that no other drug can approach, and often after numerous others have been tried and discarded as useless. Even in these days of manipulative and operative gynecology there are many young women who would prefer to be treated by medicines rather than by instrumental interference; who are very naturally frightened out of the house of a physician who suggests a digital examination as soon as a history of dysmenorrhœa has been elicited.

In the writer's hands the best effects from the use of manganese have resulted when the history is of general malaise before the flow begins, with some pain, growing rapidly worse as the flow is about to commence, and pain more or less severe during the first day. A good colour or a pale face seems to have no bearing upon the action of the drug; its action appears to be upon the nerve-centres concerned in the menstrual function, rather than upon the blood. It is no unusual thing to find a habit of dysmenorrhœa which had existed for years yield at once to the exhibition of manganese.

One word about the administration of the drug. Some women experience considerable difficulty in taking it because of a very delicate stomach, which rebels against it. For this reason it is well to begin with a small dose, say one grain at a time, and gradually increase it. The writer has given five grains three times a day during several weeks without any ill effect, but experience has taught him that three-grain doses will do as much good as a larger quantity, and that failure with that much cannot be changed to success by increasing the dosage. Often it is unnecessary to give even this much, but it may be properly pushed to this limit in doubtful cases.

Therapeutic Gazette, June, 1898.

It is not always easy to draw the line between "brain fag" and neurasthenia, that is between temporary cerebral fatigue and true ner-

vous exhaustion. Usually the two go together; but in those cases in which an intellectual worker is run down without its coming to a case of neurasthenia proper—and such cases can be counted by the thousands—what is best to be done? Dr. Romme has just made some very valuable suggestions with reference to them, and bases justly the whole treatment on hygiene and tonics. Nevertheless the key-note of treatment in all such conditions is rest. Some of Dr. Romme's suggestions are these:—The patient should go and live in the open air in the country; if he cannot, he should occupy rooms well aired, ventilated, and with full exposure to the sun. Between each period of work, covering a few hours, and especially before and after meals, he should take mild physical exercise in accordance with his tastes, such as walking, bicycling, rowing, gymnastics, open-air games, bowling, golf, croquet, lawn tennis, &c. If he cannot go out he can do gymnastics in his rooms, play billiards—in a word, seek distraction and movement. If he finds gymnastics too fatiguing he will derive benefit from carriage exercise. His meals ought to be regular and substantial, with avoidance of alcoholic drinks. Massage and douches are valuable adjuvants.

These cases are much benefited by shampooing the head every day, and then rubbing it with alcohol before drying thoroughly. It is well, however, to begin the treatment with a complete absolute rest from mental labour for several weeks before adopting Dr. Romme's suggestions. Horseback riding is a most valuable exercise and aid to re-establishment in brain fatigue. As regards tonics, there is nothing better than a phosphate combination of iron, quinine, and strychnine, alternating with a course of the glycono-phosphates.

Medical Record, July 16th, 1898.

Endocarditis Cured.—Starck cites the case of a little patient, eleven years old, who, during a second attack of rheumatism, developed an endocarditis involving the mitral valve. Treatment consisted of sodium salicylate internally and an ice-bag to the heart. In six weeks the child was cured, the heart-sounds and area being quite normal. Authentic cases of healed endocarditis following rheumatism are certainly not numerous.

American Journal of Obstetrics, July, 1898.

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ON SOME TUMOURS AND OTHER MORBID ABDOMINAL CONDITIONS.

BY

RICHARD BARWELL, F.R.C.S., &c.

THERE are in the wards a certain number of patients with various states, producing some of them enlargement of the abdomen without any defined tumour, and some of them very evident localised tumour; therefore it has appeared to me a fitting opportunity to say a few words concerning the diagnosis of these conditions. Many abdominal enlargements are perfectly easy of diagnosis, others—and here I more especially speak of distinctly localised tumours—are so extremely difficult of diagnosis that men even very largely experienced fall occasionally into errors. To apportion what I have to say among the examples now at my command, I must, before demonstrating the cases, give a rapid sketch of certain different abnormal modes in which the abdomen may enlarge. We may provisionally divide these into such as cause general tumefaction and such as produce localised tumour with more or less definite outline.

The former must be further subdivided into those due to accumulation of gases or liquids within the membranous chylopoietic viscera and such as are caused by like accumulations within the cavity of the peritoneum.*

The one may be fittingly exemplified by ascites; the other by the gas formation of acute suppurative peritonitis, ruptured gastric ulcer, or intestinal perforation.

Let me first speak of ascites. The mechanical key to the differential diagnosis between this state and many other abdominal conditions is to retain

* Note occasionally such an accumulation is limited to the lesser sac of the peritoneum. In such cases other circumstances, such, for instance, as perforation of posterior wall of the stomach, are more important than and overshadow the mere enlargement.

in the forefront of memory the fact that liquids are heavier than gases: the ascitic fluid sinks, therefore, to the lowest parts of the abdomen; while the intestines, being at least partially filled with gas, float upon it, and come into contact with the abdominal wall in front, therefore while the patient lies supine that part of the belly is resonant.

In cases where from whatever cause there may be difficulty or uncertainty of diagnosis, the patient may be placed in the erect posture, stooping, if necessary, a little forward. Resonance will in such posture be found at the top of the abdomen, dulness in the hypogastric, inguinal, and, according to the amount of fluid, higher or lower in the umbilical and lumbar regions.

Here I must diverge a little to notice the different localities of resonance in certain other cases. When a solid tumour or a fluid-containing cyst springs from, let us say, the kidney or ovary, it grows forward in the direction (usually) of least resistance until it lies in contact with some part of the anterior abdominal wall, displacing to one side or the other the intestines. Hence we find in such cases just the contrary to what is found in ascites—instead of a resonant area surrounded by dulness we find a dull one surrounded by resonance. In fact, in a typical simple case of ovarian cyst, the space where the tumour, coming in contact with the abdominal wall, produces dulness, corresponds almost precisely with the area occupied by the resonant bowels in ascites. In such cysts, more especially if simple, fluctuation is more distinct and rapid than in ascites, because the wave in them is more unimpeded and direct, not having, like that in ascites, to find its way among coils of intestine and layers of omentum or mesentery. To cysts of the kidney (pyo- or hydronephrosis) the same remarks will, up to a certain point, apply, but they are rarely central, nor do they as a rule, at least until very large, encroach upon the inguinal region.

When gas fills the peritoneal cavity the abdominal distension is smooth and even; tympany is uniform over the whole area; at first—that is to say, while the amount of distension is not very great—hepatic dulness can still be made out, but when, with further progress of the evil, the liver becomes covered by gas, the dulness is lost. If in such a case you place your flat hand on any part of the abdomen and tap lightly elsewhere on

its wall you will feel with the palm a thrill of rapid vibration, which only differs mechanically from fluctuation in that the wave length of a gas is much shorter than that of a liquid. This thrill, resonance, and distension frequently extend far back even to the very edge of the erector spinæ. Indeed, a man with suppurative synovitis, whose life I saved by abdominal incision and washing out the peritoneum with several gallons of distilled water, had such distension that on looking at his loins the abdomen was seen to bulge on each side of those muscles, they forming a wide shallow groove rather than a broad ridge, and on each side of that groove the bulging posterior wall of the abdomen was plainly tympanitic with the above described thrill.

But there is another set of cases returning a hyper-resonant note over the whole or nearly whole abdomen, and these must not be mistaken for those just described. Let me show you this woman, Mary H—, æt. 66. Nine years ago she was aware of a tumour in the right inguinal region. I do not propose to speak of the nature of this tumour nor to make any diagnosis, but only to demonstrate the difference between this form of tympanitic distension and that just described. In this case also the abdomen is greatly distended; even the umbilicus is obliterated; also it is tympanitic everywhere except over the liver. Its surface, however, is anything but smooth and even. In the left hypochondrium you see the stomach bulging forward; running across the middle is the colon. If you stoop and look along the surface, so that light reflects from it the coils of small intestines may often be seen to move (peristalsis). It is possible to obtain in selected places a thrill, as sometimes, for instance, along the line of the colon or over the stomach, but there is no free and unobstructed vibration from any and every part of the abdomen to every other part, as in peritoneal tympanitis; it is checked and impeded by the walls of the intestines, whose distension with gas you can, as just described, see. It is a case—in all probability due to partial obstruction about the lower bowel—of intestinal tympanitis. The bowels, and not the peritoneum, are distended with gas.

Before showing you a case or two of tumour within the abdomen, it would be well to explain how to distinguish such as are within that cavity from such as lie between the skin and the abdo-

minal wall. Let us suppose the doubt to arise in a patient lying on the back. If he be told to sit, or try to sit up, he will in this endeavour contract the abdominal muscles. If the tumour be outside the cavity it will at once become more prominent; if within, it will be entirely or almost entirely obscured.

Here is a woman with a tumour in the abdomen—Mary Ann N—, age 35. About eight years ago she had certain menstrual troubles, *i. e.* occasional menorrhagia alternating with amenorrhœa, and both with much pain. Three children. Much weakened, and feeling ill. Four months ago she became aware of a lump on left side of the abdomen, and had much pain and sense of weight, chiefly in epigastrium. She came into this institution on 9th, when she had a severe shivering fit, followed by fever (temperature 103°). On the next day, 10th, temperature was normal, and she remained in her usual low state of health till 15th, when she had another shivering fit, followed by temperature 103.4° .

I saw her for the second time that day while in pyrexia, her aspect, of course, different to what it is in quiescent state. The eyes were bright, face flushed in patches, skin hot and, except on forehead, dry; capillaries and veins of surface full.

Abdomen.—Inspection. Whole abdomen large, slightly more so on left side. Epigastrium protuberant and puffy; she complains of much pain there. Veins strongly marked and tortuous, but not prominent.

Palpation.—Rather more than the left half of the abdomen is occupied by a smooth solid mass. Its inner or anterior edge extends from the hypochondrium about middle of cartilage of eighth rib downward in a curved line passing two inches to the right of the umbilicus, extends to within very little of the pubes, thence into the iliac fossa, leaving a small space in the inguinal region unoccupied. No notch, as is usual in these cases, can be felt on this inner border. Behind we find its posterior edge very little in front of the sheath of the erector spinæ, running straight down from under cover of the ribs to the crest of the ilium. The whole of this area is dull, the dulness extending under the ribs as high as the sixth. This dulness is absolute on light percussion, but on pressing firmly with the pleximeter finger and striking a little harder, one hears some muffled distant reso-

nance. This tumour is movable from side to side; one can push against the posterior edge, and move the whole mass from one and a half to two inches to the right, but it can hardly be pushed back beyond the position it takes when she lies supine. Like all splenic tumours, it descends on inspiration.

She has fairly free action of the bowels. Her colour indicates some, but not marked anæmia, although it is rather turbid and opaque. The blood which I have—somewhat roughly, it is true—examined shows no decrease of red particles nor increase of leucocytes. The red globules do not run together to form piles or chains, and it seems to have a strong disinclination to coagulate.

There is little difficulty in the diagnosis of this tumour; its shape, save the rather unusual absence of the notch, its position, its mobility, its descent in inspiration, reveal to the surgeon at once the fact of enlarged spleen. Also the fact that you can feel an almost straight posterior edge excludes the possibility with approach to certainty of its origin from the kidney, and this is confirmed by absence of renal symptoms. Its shape, already mentioned as characteristic of certain splenic enlargements, is not quite that of a normal spleen, but its external surface has moulded on the abdominal wall, it therefore is convex, while the postero-inner surface is concave and fits over the intestines. The distance, resonance, and the mode of mobility, point to its being rather spread out and thin; it has no intrinsic bruit, but the movement and gurgling of underlying intestine can be heard through it. The absence of a notch is the only sign adverse to this diagnosis, but I have in the post-mortem room more than once observed that when during life no notch could be felt, the spleen in enlarging had assumed the above described shape.

The singular points in the case are that the woman has fits of ague—at wide intervals it is true, but otherwise typical; yet she has lived all her life in London, not in a damp district (Somers Town). Moreover the proportion of red and white corpuscles are not markedly, at all events, altered. If ague and spleen enlargement are the result of marsh-bred organisms, *ab extra* in the circulation, whence did this woman acquire them, and how does she get so big a spleen without some evident blood change? I think I must ask Dr. Hopkins to let me make or rather procure

an expert search for plasmodiæ and crescent bodies.

As to surgical treatment. Removal of so large a tumour as this would require a very wide incision into the abdomen, not that such is of much importance now-a-days; the vessels supplying the huge gland are in all probability very large, augmented in number, and would probably be difficult to deal with. The records of splenectomy are not very encouraging. If, however, the operation be undertaken it must be done before leukæmia sets in. Forty-nine removals of leukæmic spleen have every one of them proved fatal, whereas in simple hypertrophy the mortality is 78 per cent., which shows that even in the less unfavorable cases such operation is very formidable. At all events medicinal treatment should be pushed to the full before surgical interference is considered, especially as those who have survived do not by any means regain health, but become affected by myxœdema, and, as far as I know, live but a short time. Whether in such the continual administration of thyroid extract commenced immediately after operation might prevent or retard such changes has not yet been proved.

Bridget W—, æt. 43, has had two living children, and up to February, 1897, had fair health. About that time she became pregnant. Nothing remarkable in the gestation except that she felt more "awkward." Of course this is not valuable evidence, save in its negative sense. In beginning of September hæmorrhage came on, and continuing throughout that month it was thought necessary to induce premature labour, which was effected on October 3rd last, and the bleeding ceased. Her abdomen remained rather large, and soon began to increase. On the 8th January she was attacked with severe pain in the right side, extending to the knee, and she was very faint. There was in the right side a perfectly hard, unyielding tumour with much the same dimensions as now; in a few days it pointed about two inches behind the mid-axillary line, and on the 8th, *i. e.* ten days ago, it was opened a little in front and below tip of the twelfth rib. A quantity of thick flocculent pus was evacuated; the finger, passed in, entered a wide cavity, and in front was only separated by the abdominal walls from a hand placed there. The wall of this cavity wherever the finger could touch it was slightly rough, calcareous, with egg-shell

crackling. A large drainage-tube was kept in the opening, and the whole side covered with antiseptic dressing and peat-wool.

Her present state is this: she is feeble and very thin, without high temperature; on the right side of the abdomen is to be felt a hard, smooth-surfaced tumour, extending from about the middle of Poupart's ligament to the hypochondrium; its limit internally is in a curved line from the ninth costal cartilage, missing the umbilicus by two inches, and terminating a little outside the middle of Poupart's ligament. Behind no edge is to be felt; it loses itself under the lumbar muscles. All this area is quite dull to percussion, the dulness extending under the hypochondrium as high as the fifth rib. Of course some portion of this upper part is hepatic. The tumour is not movable in any direction. At present the discharge is sero-purulent, somewhat deeply stained with blood; it is odourless, and contains neither urine nor cholesterine.

Examination *per vaginam* reveals only that the right fornix is slightly depressed and appears a little thickened; the uterus is small and somewhat flaccid. The rectum is largely ballooned; the finger lies loose in the wide cavity, and touches nothing unless with quite a little effort. No stricture or compressed portion is within reach, though the condition is such as is found when stenosis of the gut lies beyond.

The diagnosis of this case is a little difficult, but can be made out with very tolerable precision if we collate the negative with the positive signs and with the history. The absence of urinary and of fæcal odour, though not of themselves absolutely excluding the idea of intestinal abscess or of pyonephrosis, render them highly improbable; neither is there pus in the urine or the stools; constipation is not severe.

If, however, we consider the fact that this cavity has calcareous walls—a condition which does not occur in mere inflammatory abscess—we shall be driven to the history for the solution. Hæmorrhage supervening in the seventh month of pregnancy was checked by the induction of premature parturition, but she did not regain her proper size, and in the next few weeks got somewhat larger, till on January 8th sudden and severe pain with faintness supervened. Then came evidence of swelling and "lump" in the right side. Now in the later stages of a disease it is always a difficult matter to

read backward into a case the meaning of present symptoms, but the calcareous walls give a clue which facilitates this to such degree that I have little hesitation in pronouncing this to be a case of ectopic pregnancy occurring simultaneously with a normal one. Such cases are rare, and perhaps even more rare are such ectopic pregnancies that go on to full term. On other points we are reduced to mere surmise. The ectopic foetus, implanted probably in the ampulla, may have been dead long before January 8th, and the sort of crisis that then occurred may have been due to and simultaneous with inflammation in the gestation sac; or another solution may be found, viz. that the ectopic gestation was the result of superfœtation, and the advent of bleeding at the seventh month of pregnancy the result of the two pregnancies interfering with the progress of both. But these two last considerations are, as above said, mere surmise; the one and probably only point on which we may feel secure is that the tumour we have here is an ectopic pregnancy gestation sac which has ruptured, the present cavity being formed in part by a portion of the peritoneum shut off from the general cavity by adhesions. Whatever remains of the foetus will have undergone some change—one of three—viz. mummification, adipocere, or lithopædion. Of these the most probable is the last one, since the condition of the cavity wall indicates decided tendency to calcification.

Probable future and treatment.—Dr. Hopkins has opened and partially drained this cavity; but the walls being stiffened with calcareous matter cannot contract, and there is, taking my diagnosis as correct, a changed foetus in the sac; therefore suppuration, with probably enough hæmorrhage to stain the secretion, will go on. Even if this be kept quite sweet, exhaustion must in time supervene, and will kill the patient. But it will be very difficult, especially in such proximity to the colon, to prevent putrefaction, and with this either pyæmia or septicæmia will set in. Of course it is far from my intention to dictate to the excellent medical officer of this institution any line of treatment, but I may say that I think he will find it necessary to enlarge that opening, which can be done without involving the peritoneum, pass his hand into the cavity and remove the foetal remains, then with gentle touches here and there destroy the continuity of the calcareous dome-like walls suffi-

ciently to allow them to fall together and close the cavity. Of course aseptic precautions must be very rigidly followed, and the cavity be frequently washed out with a mild but efficient antiseptic lotion.

Before closing it may be well to give a sketch—a mere outline—of the chief tumours and of their characters. All tumours, including cysts and abscess of the liver, cause enlargement of the glands. Syphilis, cirrhosis, inflammation, congestion, amyloid and fatty degenerations have the same effect: thus mere increase of hepatic dulness is of itself no proof of the existence of tumour; but should a definite lump be felt in the region of the liver and the gland be much bigger than normal, that tumour or cyst is large in proportion to the hepatic increase. It must be remembered that the left hepatic lobe passes a good way beyond the middle line, and there may be some difficulty in distinguishing growths on or in the gland from pyloric, duodenal, or pancreatic tumours. The general symptoms will, however, greatly aid the diagnosis; moreover, these last, which I shall not further mention, usually make their appearance lower on the abdominal wall. Solid tumours of this part are cancer, generally secondary, gummata, hydatid, and abscess. This last, if not traumatic, is nearly always due to acute dysentery or other such diseases as cause intestinal ulceration. Of hydatids I have a few words to say: when they approach or originate so near the surface as to be palpable, their globular shape and elasticity render their diagnosis pretty accurate. A fine trocar drawing off the fluid will make it certain. Of all parts of the body the liver is the favourite habitat of the parasite, and from my experience I should say that the left is a more frequent site than the right lobe, yet this may be due to merely a fortuitous succession of cases. A very common and proper mode of treatment is to evacuate the hydatid fluid and to replace it by injection of iodine or other germicide, with the hope of killing the animal, and when the cyst is single or nearly so, this often succeeds; but if that object fail two or three times it is unwise to go on, and the proper treatment is incision. Some little time ago I read a paper at the Royal Medical and Churgical Society ('Lancet,' 1887, vol. 1, pp. 217, 340,) on the incision in two stages of hepatic cysts. Having opened the abdomen and stitched the parasite, without perforating

its wall, to the peritoneum, I waited till the third day and then incised. With the fluid there came away a great number of daughter cysts, and in the next few days very many more—at least 100 in all; lastly, the wall of the primary cyst made exit, and the wound healed. Let me just add that this result shows that a hydatid containing many progeny cannot be cured by injection, since even were a few secondary cysts punctured and killed, there must still be left a superabundance of living ones to multiply the parasitic race. I believe myself to be the first surgeon who has proposed and employed this mode of dealing with hydatids.

Together with tumours of the liver we must take enlargements of the gall-bladder, due sometimes to mere distension with bile, to empyema, to hydrops, to gall-stones, &c. Enlarged, this receptacle forms a tumour lying just below the cartilage of the ninth or tenth rib, which on its still further increase extends downwards, forming an elongated ovoid tumour with the long axis directed a little obliquely towards the umbilicus. The diagnosis is not always easy, for movable kidney frequently presents itself in this locality, and descends on inspiration, being pressed down by and with the liver. Even an exploratory incision does not invariably lead to correct judgment; as error has been made even by a much practised laparotomist. (See Osler, "On Abdominal Tumours," 'New York Medical Journal,' vol. 1x, p. 65). Nevertheless, with due care such mistakes can be avoided. The operation of cholecystotomy, neither severe nor arduous, in the generality of cases may under certain circumstances become very difficult. In some cases it is impracticable to clear the duct of an impacted stone of papilloma, epithelioma, or other obstruction when a communication direct from the bladder to the intestine must be established (cholecystenterostomy). Occasionally when the abdomen is open the tumour may be seen to be malignant, when the parietes had better be sutured again.

Of kidney tumours, some are solid, some cystic; they make their appearance in the lumbar, lower part of the hypochondriac, and the outer portions of umbilical regions, occupying more or less of this area according to their size. The renal region behind is more or less full; indeed, it becomes protuberant when the tumour is large. A hand placed on the mass in front can press the organ

and excrescence back into the other hand at the loin, and this latter again propel it forward against the anterior abdominal wall. This sort of *ballotement*, if I may so call it, is very characteristic. Yet there are other distinguishing points; for instance, it differentiates itself if on the left side from an hypertrophied spleen by not descending with inspiration, and on the right in the same way from hepatic tumours. The globular tumour when felt from the front gives an idea of bulk and thickness (different to splenic, generally thin, tumours). On the left side one may frequently—*i. e.* when the colon contains gas—find resonance in front of its outer half, while on the right side one may elicit that note from the ascending colon on the inner side of the tumour, which has pushed the gut over towards the middle line. Often one may find a rope-like elevation crossing the tumour, which at times may be resonant, at other times in the same case dull, according as the coil of small intestine, the rope-like eminence, contains gas or solid matter. Movable or wandering kidney is much more frequent in women than in men—the organ may be in almost any part of the abdomen. I have found such close under the liver or just below the right costal cartilages, and also in either inguinal region. It may be anywhere between these four points. The loin whence the kidney has emigrated feels empty and hollow. By placing the patient in a sitting position the gland can be pressed back by a hand on the abdomen, and can be felt to fall into its place by the other hand at the loin. If bandages or truss-like instruments fail to alleviate the distress caused by this condition, the gland may be sewn into its proper position. The operation, not altogether an easy one, and least so in fat persons whose last rib comes close to the iliac crest, is, however, one in which I have had no little success.

I should have liked to speak to you of tumours connected with the intestines, especially of the lump caused by intussusception and volvulus, but having already somewhat overstepped my limits, and given you as much concentrated matter as may be digestible at one meal, I will, with your permission, stop at this point.

ON PRACTICAL POINTS IN PERCUSSION AND AUSCULTATION, AND IN THE PHYSICAL EXAMINATION OF THE CHEST.

ABSTRACT OF LECTURES TO ADVANCED CLINICAL STUDENTS

BY

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II.

ON SOME PRACTICAL POINTS IN CONNECTION WITH INSPECTION, PALPATION, AND PERCUSSION OF THE CHEST.

1. *Inspection.*

This is the simplest and the easiest of all our methods; and yet it rarely receives the attention which it deserves. All that you are required to do is to look and to see. Beyond this there is no technical refinement that I can suggest. That which you doubtless have already done well you should endeavour to do still better. Lend to this work the best use of your eyes, so keen and successful in matters of sport; and remember that in order to see everything that is to be seen you must look very intently. Make it an invariable rule to turn to the best advantage any light you can get. The first requisite for diagnosis in general, and the essence of inspection, is to place your patient in full and direct light. Attention to this may make all the difference in your future success as practitioners; and I can only repeat to you the advice which I once received from a great clinical master, the late Sir George Paget, "never stand in your own light."

Then as to sighting. It is hardly for me to teach you how to shoot. Apply the principle you know so well. Sighting patients' chests means raising and lowering your head to the level of the surface you wish to inspect. This implies a little trouble, but is worth it, as you will not fail to perceive from your improved results. In other words, the patient's position should govern your own.

(1) *Sighting from above* is an excellent method when the patient is seated. As you stand behind the patient you command a view of the front and of the back of the chest, and if the light is good you will detect any fine differences in shape and in movement. At the same time you will have an opportunity of noticing the configuration of the back and the sagittal diameter of the right and of the left side of the chest.

(2) *Sighting from below* is easiest with the patient standing, when by bending the knees and looking up from the front you may get an excellent view, particularly when working with a top light. This performance is slightly trying for the observer, but you will think nothing of this inconvenience when in earnest to detect the indications which may decide in a difficult case as to the presence or absence of a severe disease such as phthisis.

(3) *Sighting from the side* presents no awkwardness where the patient is standing or sitting, and I need say no more about it.

(4) *Horizontal sighting* is requisite where the patient is lying in bed. You have no choice but to stoop to the level of the object for inspection. This is the method which you will find most useful in the wards; it applies not only to the inspection of the chest, but to an examination of the abdominal viscera also. If you will only give sufficient attention to these practical measures you may soon outdo your teachers in the accuracy of your observations.

2. *Palpation.*

This large subject includes two great divisions, superficial and deep palpation.

(1) *Superficial palpation.*—Here, again, in your advanced work you will find little to add to the elementary method which you have all practised; and the only suggestion I can offer refers to increased care and attention in its performance, so that you may learn to feel keenly. I would merely remind you of the advantage to be gained from an intelligent use of the different parts of the hand, the tactile appreciation of which is far from being uniform; but to this point I must refer later on.

Three distinct objects belong to superficial palpation:

- (i) Palpation for characters of surface.
- (ii) Palpation for vibrations.
- (iii) Palpation for movements.

Simple palpation is that which in the blind has to do duty for sight; with us it may be regarded as "supplementing" sight. The thickness and quality of the skin, its swellings, œdema, or loss of elasticity, its subcutaneous fat or nodules, any abnormal state of the muscles or superficial changes in the bones, or any malformation giving rise to irregular prominences or depressions of the thoracic skeleton, and in particular the rachitic deformities and the so-called "rosary,"—all this falls within the scope of simple palpation, which also enables us to determine the situation of the ribs and of the costal arch, of the sternal notches, and of the xiphoid appendix. For its successful performance you should not depend exclusively upon the tips of the fingers, but often use the flat of the hand as well.

Palpation for thrills is not always a purely passive operation. Some thrills are obvious; others need for their detection the "tactus eruditus." The hand must therefore be applied with a variety of pressures, or you may fail to perceive the finer thrills.

By applying the whole hand you bring into play all its sensitive areas, and particularly the thenar, the hypothenar, and the post-digital eminences of the palm. If the thrill be of large extent you will feel more of it; if small, as in the case of most heart thrills, its perception localised to a limited portion of the hand will be rendered more striking by contrast with the absence of any sensation of thrill from the rest of the palm.

When testing for *vocal fremitus* save time by using both hands, each hand being used for the corresponding side of the chest. By alternately applying and raising one hand, and then the other hand, you will at once detect any marked difference; but if necessary you may confirm your results by using the same hand in succession to each of the two sides.

In ascertaining the level of any change in the vocal fremitus the *ulnar border* of the palm is often useful; it is convenient both in respect of its shape and of its tactile sensitiveness. The flat of the hand having been applied to the surface to be examined, its outer border is gradually raised till the hand hinges on its ulnar border, and whilst the patient is still counting it may be, if necessary, brought down once more into full contact with the chest. The precise level at

which the alteration in the vocal fremitus occurs can in that way be determined with accuracy.

Again, a careful graduation of the *pressure* of the hand is all-important for the detection of obscure pulsations, whether cardiac or aneurysmal, and for the latter when deep-seated, firm pressure is sometimes needed. Venous thrills, on the contrary, require very light pressure.

Palpation for movements should always be called to aid to confirm the results previously obtained by inspection. The diffused cardiac or aneurysmal impulses, the systolic movements communicated to the interspaces and to the ribs, as, for instance, to the twelfth rib, the cardiac systolic succussion of the thoracic surface, &c., are instances in point.

Palpation has this advantage, that we can compare both sides simultaneously, whilst sight can only deal with them in succession, and having to glance from one to the other loses continuity of sensation. Another advantage is that by palpation we can to some extent influence and govern the respiratory movements, and thus assist our observation. In this connection I can recommend to you the following practical method.

Resisted respiratory movement.—A clear notion of the respiratory energy of the patient and of his available range of respiratory movement is an important element of diagnosis. Some patients are very shallow breathers whilst under examination, and we may be left in doubt as to their respiratory capacity. A definite conclusion may be arrived at by the bimanual application of combined palpation and pressure. One hand is placed on the back, below the scapula, the other on the pectoral region of the same side, and gentle but firm pressure is made by both hands during expiration, with a view to encouraging a complete emptying of the chest. At the end of expiration the pressure is relaxed slightly, and the patient is directed to inspire fully. Whilst he is drawing his breath the pressure is to be kept up, and its degree is to be carefully adjusted to the inspiratory effort which has been called forth by the resistance, the object being not to prevent the thoracic expansion, but to gauge the activity and the range of the full inspiration which is thus elicited. The mobility of the axillary regions is then tested in the same manner, by grasping between the hands the two sides of the chest. By this means you will be able, after a little practice, to distinguish functional

laziness of respiration from the limitation of breathing which is due to structural disease.

(2) *Deep palpation* is a method the full development of which we owe to Dr. Maguire. It had long been applied to the abdomen, but he has shown us that it may be applied to the thoracic organs as well, and that under favourable circumstances it may even be possible to determine by its means the boundaries of the viscera contained within the chest. I will now demonstrate to you in a patient whose heart has been displaced to an extent which I have not yet ascertained that this form of palpation can yield excellent results.*

This method is an odd one in many ways. It implies *deep* palpation, and this can only be carried out if the pressure be gentle and soft as well as firm. My advice would be that you should practice at first upon forgiving friends rather than upon your enemies. Select for your early attempts juvenile subjects with exceedingly pliable chests. With them you will get at the organs more readily than in larger chests with thick coverings of muscle or fat. As your touch becomes more educated and lighter it will be less resented; and you will acquire by degrees the delicacy of tactile feeling which is the essential point in this method.

3. *Percussion.*

There are a few technical points in percussion due attention to which makes all the difference between the apprentice and the artist. These include a full mastery of the light and of the heavy stroke, and their opportune employment where each of them is called for; an intelligent use of sufficient pressure with the left or pleximetric finger,—this pressure also needing variation; a careful training of the ear as well as of the touch, upon which we depend for clear notions as to the results of percussion; and a due appreciation of the local anatomical peculiarities of the several parts of the chest.

Single Stroke Percussion.

The sure sign of an experienced percussor is that he rarely gives a "double knock." If strokes

must be repeated, a sufficient interval should intervene between them. The beginner, who is seldom satisfied with less than three or four, does not realise that the last stroke in a rapid series is the only one that can convey a definite impression to the ear. He should be warned against the habit, and reminded that one stroke instead of two on every occasion during a lifetime will mean a vast saving of time, of discomfort to his patients, and of damage to his own finger. A complete examination of the chest, even with single strokes, means many blows. In examining the back considerable strength has to be used by both hands, and it is here that the finger will suffer severely from any unnecessary rapping.

Freehand or swinging percussion from the wrist and elbow, and fine percussion from the wrist.

You will bear with me if I touch upon these and other elementary details which are fundamental. We never percuss from the shoulder unless suffering from an ankylosed elbow or a stiff wrist. The use of the elbow is combined with that of the wrist in rough percussion; but fine percussion is to be made exclusively from the wrist. The first method is the "swinging," "freehand" (or rather perhaps "freearm") percussion. The second is the fine "local" percussion—two totally distinct methods.

By the "swinging percussion" we draw the sound from as far and as wide as possible. Fine percussion carefully avoids doing this, and seeks to analyse the slightest local shades of resonance.

Freehand percussion from the wrist and elbow is therefore well suited for a preliminary exploration of the chest, particularly if apparently sound, and will be of use in rapidly detecting coarse changes, such as the presence of fluid or of considerable consolidation. For the detection of limited and slight changes *fine percussion*, to be practised exclusively from the wrist, is indispensable; and this is also essential for the correct mapping out of organs. The pleximeter is fortunately not much in favour in the schools, and finger percussion is therefore not deprived of the attention which it needs. But fine finger percussion is a much more difficult performance than the use of the pleximeter, and consequently for the purposes of practice, where definite results must be obtained rapidly, a pleximeter such as Sansom's is of special service. Since,

* The case was one of cardiac displacement to the right, resulting from pleural adhesion and contraction of the lung. The boundary which was determined by deep palpation was found to coincide exactly with the tracing subsequently obtained by fine pleximetric percussion.

as previously stated, it picks out and intensifies any slight dulness, no dulness escapes its investigation, and its shape is such that the outline of the dulness can be worked out to a small fraction of an inch, although this statement is often received with incredulity by those who have never acquired sufficient skill in the method. The fine finger percussion method affords us the possibility of so regulating the action of the percussing hand that the successive blows are uniform. The weight of the hand being always the same, provided, of course, it be used passively, the rest is merely a question of length of "drop."

A good way to acquire the necessary skill is to practise percussion with the right elbow and forearm resting on the table, whilst the hand and wrist project beyond its edge. In this way an ample range of vertical movement of the wrist can be acquired. For the study of fine percussion the hand and wrist should be on the table, and the wrist should remain in contact with it.

On Factitious Percussion Notes and the Use of the Left Hand.

Two of you have just percussed the same spot on the patient's chest; the first percussion was almost inaudible, the second we have heard plainly. You should all endeavour to obtain a good, full-sounding percussion, and you may all succeed in doing so, for the whole secret lies in the use of the left hand.

It is possible to apply the left finger so firmly as to stifle vibrations, but the common mistake is the opposite one. Want of tone usually does not mean the lack in you of that quality described as "touch" in the pianist, but simply the neglect of that amount of pressure which is necessary to condense the media and render them more pleximetric. A well-adjusted pressure not only gives a louder sound, but favours also your perception of the tactile vibrations.

Factitious results are clearly, then, a possible fallacy. Since different pressures give so much difference in the note, everything would seem to depend upon the percussor. It must be confessed that such is the case. If sufficiently skilled he can manipulate the notes in a manner calculated to shake our faith in the method; and this is one of the reasons why each observer prefers to trust to his own percussion.

The best remedy against being misled into unconsciously manufactured results is to be alive to their possibility and to their mode of production; in other words, to acquire the necessary skill for producing them at will. When he has reached this stage the percussor is an expert, and his ability to produce deceptive effects is his best guarantee that he will not be himself deceived.

Direct Percussion as applied to the Clavicle.

Direct or *immediate* percussion without the intervention of either pleximeter or finger, can only be applied to subcutaneous bony surfaces. Of these the chest presents but two, in addition to the spinous processes of the vertebræ, viz. the clavicle and the outer portions of the scapular spine; and the clavicle is the only one which is commonly percussed in this way. I shall devote to this subject my concluding remarks.

The clavicle belongs to those surfaces which I shall describe in my next lecture as the specialised percussion areas. The length and the elasticity of the clavicle are important peculiarities, but they are not the only ones. This bone spans areas possessing very different percussion values between the highly resonant tracheal region and the exclusively solid masses at the tip of the shoulder, and it crosses only a small width of lung in its second sternal quarter. The left apex, as is well known, is even narrower than the right; in spite of this, however, there is in health a practical equality in the results of percussion of the right and of the left clavicle, but in both alike the general result of the variety in the underlying material is that no two spots in the clavicle afford the same note on percussion. It will be found by successive percussions conducted from the middle line to the shoulder, that as we proceed outwards the pitch of the note rapidly rises; and a fine ear will detect between the deep-toned tracheal percussion note of the sternal end and the high-pitched note of the shoulder end a considerable musical interval.

Obviously, then, no conclusions can be drawn from a comparative percussion of the right and of the left clavicle, unless this be conducted on both sides at exactly symmetrical spots. This suggests a method which is both reliable in diagnosis and practically useful—that of bilateral and alternating percussion with the right and the left hand, the percussor standing exactly opposite the patient.

The method is shown in the illustration, which I owe to the kindness and skill of Mr. H. G. Drake-Brockman.

Fig. 1.—Showing the alternating percussion of symmetrical spots on the left and on the right clavicle. The percussion should be made from the front, not as in the figure from behind.

In this way the right and the left percussing finger can alternately sound identical segments of the two clavicles, and demonstrate any differences between them with the least expenditure of time. Proficiency will easily be acquired even by those who are not ambidextrous.

This same principle of symmetry in percussion is applicable elsewhere, but owing to the one-sided position of some of the thoracic organs it is applicable only to the upper front and back and to the axillary regions of the chest.

Since the apex of the lung underlies so small a proportion of the length of the clavicle, the percussion of its outer half might seem superfluous. Yet it is constantly practised, and not without profit, because on the one hand the clavicle is a pleximeter readily transmitting vibrations, and on the other hand its outer extremity is subject on

both sides to identical dulling agencies, whereas its inner half is influenced by the strong resonance of the trachea, and by that of its divisions which are not symmetrical. It follows that any marked difference in the note obtained over the two outer halves will be a reliable source of information, although the condition producing the note is at a distance from the point percussed. Indeed, cases may be met with in which more definite information might be obtained from the percussion of the distant part of the clavicle than of that immediately above the lung. A similar value attaches to the percussion of the outer part of the scapular spine, which is a long way external to the pulmonary apex.

WHAT detrimental effects, if any, does the excessive formation and absorption of indol entail upon the human organism? The effects of indol upon the human subject, taken in conjunction with a study of the clinical conditions in which the indoxyl reaction is markedly increased, justify us in believing that prolonged and excessive indol absorption is capable of causing headache, especially frontal headache, abnormal cephalic sensations, and indisposition for mental and physical exertion. The latter condition, if prolonged, may perhaps form the basis of a neurasthenic state. Although this is as far as we can safely go with our present knowledge, it is not at all unlikely that further investigation will enable us to detect other effects of excessive indol absorption. That the individual susceptibility varies much seems clear from the results of experiment, and it is likely that this difference in susceptibility relates both to the intensity and to the character of the influence which is exerted. There is no doubt that some robust persons may habitually excrete a large amount of indoxyl-potassium sulphate without showing definite evidence of derangement of health, but these cases are certainly exceptional. While, therefore, we cannot regard indol as an indifferent substance in the human organism, we cannot regard it as ordinarily exerting highly toxic effects, even when it is absorbed in unusually large amounts. This conclusion accords with what one might on *a priori* grounds expect in the case of a normal decomposition product of proteid food.

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ON IDIOGLOSSIA.

With Special Reference to a Case in which it was associated with Pseudo-hypertrophic Paralysis.

BY

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IN TWO PARTS.—PART I.

THE name Idioglossia was given by Dr. Hale White and Mr. Golding-Bird ('Med.-Chir. Trans.,' March 10th, 1891) to a defective condition of speech so peculiar as to give the impression that the speaker used a language of his own. They exhibited to the Royal Medical and Chirurgical Society two cases of the kind occurring in brothers aged 9 and 10½ years. At the same meeting of the Society, Dr. Frederic Taylor reported another instance of idioglossia in a boy aged 8½ years. The late Dr. Hadden had previously given a very full account of three similar cases, all males, aged 11, 7, and 4 years respectively, in the 'Journal of Mental Science' for January, 1891.

Professor Wyllie met with two imbeciles at the Larbert Institution, who presented the same defect of speech. One was a boy aged 8, the other a girl of 20 (Wyllie, 'Disorders of Speech,' 1894, p. 122). Dr. W. S. Colman reported two cases in boys aged 6, and had met with six others ('Lancet,' vol. i, 1895, p. 1419). The present writer has seen a number of children and adults in whom the defect existed in a minor degree, and two children in whom it was well marked.

The first of the latter was a girl aged 7, of whose case unfortunately the notes are incomplete; * the second forms the subject of this paper.

* This girl, whose alphabet is given in phonetic form on p. 315, was not unintelligent; she could read words of one or two syllables, and could write fairly well. Her speech presented most of the characteristics presently to be described. She also showed in a marked degree the tendency to "mind-wandering" so common in this affection. Whilst her attention could be engaged she could repeat most words correctly at dictation, but she soon looked vacantly round the room, and her speech became gibberish, although she still continued to mechanically utter words after me.

This patient, a bright and intelligent-looking boy aged 7½, was admitted to the Hospital for Epilepsy and Paralysis, Regent's Park, in February, 1898, for the treatment of pseudo-hypertrophic paralysis. He had been forward as a baby, walked at fifteen months, and seemed strong on his legs until two years ago, when his gait became awkward, and he began to fall frequently, whilst the calves of his legs gradually grew in size. An elder brother of his began to lose the use of his legs at nine or ten. His calves were at first swollen, but later became wasted. His weakness increased until he was unable to stand or walk. Subsequently his upper extremities became affected, and he died suddenly at the age of fifteen. He was treated for Duchenne's palsy at Guy's Hospital about six years ago. No other members of the family are known to have suffered from this disease.

In the present patient the lower extremities are chiefly affected. The gastrocnemii are very large and firm; the vasti externi, especially at the left side, are larger and more prominent than usual. The glutei seem unaffected. There is no enlargement of the scapular muscles or of the pectorales. The infra-spinati are perhaps slightly wasted.* The triceps muscle on both sides and also the muscles of the forearms are a little increased in size.

He has slight lordosis, and leans backwards with his shoulders well behind the line of gravity as he stands. His unsteady gait, with legs wide apart, and his method of uprising from the sitting position, are typical of pseudo-hypertrophic paralysis.

It is not my purpose to discuss pseudo-hypertrophic paralysis in this paper, or to do more than mention that it is here associated with idioglossia. The association is probably quite accidental, but it must be mentioned that the brother who died of Duchenne's palsy is said to have spoken exactly as the present patient does until shortly before death, when his speech became clear and distinct. I cannot vouch for the truth of this. A sister of the patients, aged five or six, is said to speak more indistinctly than either of her brothers, but as yet she has shown no signs of pseudo-hypertrophic paralysis.

In order to give a general idea of the defect of

* Three months later these muscles were found to be decidedly enlarged.

articulation under which he labours, the alphabet as phonetically pronounced by him may first be considered.

The alphabet, perhaps, does not best illustrate his speech imperfections, because the sounds of the letters when uttered singly are not the same as when combined to form words; but it is difficult to devise a better scheme for demonstrating his defects, and at least the knowledge of the particular letters which he mispronounces has been of service in rendering his speech intelligible. At first he could not be understood at all, but this was largely due to slurring and indistinctness of utterance. When he could be persuaded to raise his voice, and speak slowly and plainly, the fundamental defects of pronunciation were easily recognised, and his language could then be interpreted.

ALPHABET AS PRONOUNCED BY H. P.

A—ah, B—bee, C—tee or dee, D—dee or tee, E—ē, F—āā, G—d, H—ah, I—ah, J—day, K—kay, L—āā, M—em (sometimes, but more often āhī), N—air, N—ō, P—p, Q—koo, R—ah, S—āā, T—t, U—ōō or dōā, V—bee, W—dādo, X—ak, Y—bāā, Z—dair.

Of the vowel sounds, A alone is always sounded as in "ah," but the sound "ā" appears for F, L, and S, all pronounced as "āā," also for Y (bāā) and J (day). E and O have their natural sounds. U is ōō or dōā, I is ah. Y again has a vowel sound, āā, preceded by the sound of b or d. "Yes" becomes dāā or bāā (ā as in day).

Of the consonants, B, D, P, T, K, are correctly given, except that D is sometimes pronounced t. But they are substituted for other consonants. C becomes d or t, G becomes d. He says "do" for "go," but oddly enough the G in "God" is pronounced with perfect clearness.*

P and B appear as initials, but not as terminals.

Sibilants, such as S or C soft or X, have no place in his spoken language, but are represented by the sound t, or by vowel sounds. Thus "sheep" is rendered tēē; "slipper," tēēēr; "stool," tooā; "biscuit," bickēē; "stocking," tockēē.

Yet he imitates the sound of hissing quite easily

when shown how to produce it, although he does not use it in speech.

"L" is represented by vowel sounds āā, sometimes with the addition of consonants d or p. Thus lion becomes "dāi," fly is "pāi" (a as in "ah").

Nevertheless he can produce the sound of "L" when at the end of a word, for dog becomes "dol."

Similarly M is usually represented by the vowel sound "ahi," but by an effort, and when instructed how to frame his lips for the purpose, he can pronounce it perfectly. N also has a vowel sound, but he can say "no" quite clearly.

"Pinafore" becomes "pēēafōh." Here the sound of "f" is produced, although if asked to say the letter "f" he again emits a vowel sound. R is not burred, but pronounced "ah;" needless to say he does not aspirate the letter "h."

To sum up, his vocabulary consists almost entirely of consonant sounds B, P, D, T, K, and of various vowel sounds.

He does not make use of the labio-dentals F, V, at the beginning of words, nor of the linguo-dentals Th, S, Z, and C (soft).

Of the anterior linguo-palatals, T and D are given clearly, but substituted for each other. Sh and Zh and R are absent. Ll and L are used only when terminals. N is variable.

Of the posterior linguo-palatals K and Ch (hard) appear. G is variable, but is usually D. Ng is absent.

Of the labials, P and B are freely used when initials, but not as terminals. W has only a D sound. M is variable.

Mental condition.—He looks and is intelligent, but owing to his physical infirmity his education has been much neglected. He cannot read, but knows his letters, and can count up to 100.

He is able to form most of the letters of the alphabet correctly, but there are some capitals, such as K, M, which he cannot draw. If asked to copy one of these from a print held a little way off, he carefully traces the printed letter with his pencil, as if to fix it in his memory, then hurries to his slate to reproduce it; by this time, however, he seems to have forgotten its appearance, and he can only draw a hieroglyphic which does not in the least resemble the letter in question. But if the copy is held so that he can keep his eye upon it and his pencil at the same time, he imitates it fairly well.

* In explanation it may be mentioned that he is a very devout little person. Doubtless more pains have been taken to make him pronounce the name of the Deity correctly than in the case of other words.

From this it would seem that his *visual memory* is defective.

In confirmation of this may be added the fact already mentioned, that if he can be made to watch a speaker's face and frame his lips and tongue as he (the speaker) does, he is able to produce sounds such as those of M, S, and F, which he does not ordinarily use. But if his attention wanders, and he looks away (as he usually does after a few moments), the sounds are no longer produced correctly, although he goes on trying to imitate the speaker.

There are, I believe, no elementary sounds of letters, or of letters combined in words, which he cannot utter if his attention can be gained sufficiently to make him observe the mechanics of their production. Yet he does not seem to know whether he has uttered them correctly or not.

He has no physical deformity of any part of his vocal apparatus. His fauces, palate, teeth, and tongue are normal.

Hence it may be concluded—

(1) That he has no defect in his motor apparatus of speech.

(2) That his visual memory is bad, in the sense that he does not remember the mechanism for producing certain sounds unless aided by his eye.

(3) That want of attention and mental concentration are largely concerned in perpetuating his faulty articulation. The matron of the hospital (Miss Oldham) states that when he is in bed, preparing to go to sleep, he can be made to say words which he seems unable to pronounce when up and about. Perhaps his mind is less actively employed and his attention less liable to be distracted at these times than during the day. Parrots are more easily taught to speak when quietly roosting than when engaged in clambering about their cages and otherwise diverting their minds after the manner of parrots. Yet parrots learn to speak by "ear," and in this respect he differs from them.

(4) As there is no physical deformity which prevents him from producing all the sounds necessary for intelligible speech, and as he is obviously not conscious whether he produces such sounds correctly or otherwise, we must conclude that the defect is not in his motor vocal apparatus, but in his ear.

It is not that he is deaf, but that he lacks the

power of discriminating shades of difference between sounds.

In favour of the view that audition rather than articulation is at fault is that the child has but little ear for music. At first it seemed that he had none at all. He made no attempt to imitate the sound of a note when struck on the piano or sung to him. He had a large repertoire of nursery rhymes, but if asked to sing one to a simple well-known tune he merely recited it in his usual fashion, without any suggestion of singing. He said that he did not know any songs or hymn tunes, and this appeared at first to be the case. But he was subsequently overheard crooning to himself sounds which distantly resembled those of a carol called "When shepherds watched their flocks by night." The carol, however, was recognised more by the child's rendering of its rhythm than of its melody, for he showed the utmost impartiality as to key, which he changed almost at every bar when singing alone.

When the accompaniment was played for him in F he sang more or less in C. But on transposing it to C and various other keys his obligato was *ad lib.* to a generous extent. He kept time fairly correctly, but was never by any chance within a tone or so of the note desired. Sometimes, quite by accident, he would sing nearly a third below the accompaniment. Evidently the musical sense is singularly wanting in his case. It is true that the singing voice of children at his age is commonly tuneless owing to ataxy of their vocal apparatus from want of training. Their musical intervals are often faulty, and they may sing sharp or flat. But for any one with a trace of musical ear it is an extremely difficult feat to sing in C, for instance, to an accompaniment played in so remote a key as F. It was subsequently ascertained that great pains had been taken by his family to teach him this particular carol, and that he had never shown any sign of acquiring another tune.

Auditory memory.—It is not easy to decide whether his auditory memory, or memory for sounds, is at fault as well as his visual memory, or memory for the seen mechanism of sound production. But it may be that this is so, and that he neither remembers sounds nor their mode of production, nor discriminates between them.

Children learn to speak by imitation. They imitate sounds which they hear, and movements

of the lips, tongue, and mouth which they see. Setting aside the difficulties in controlling and co-ordinating their vocal apparatus which have first to be overcome, the power of speech is developed quickly or slowly in accordance with the accuracy of perception, both visual and auditory, which may be present. The imitative faculty is not possessed by all in the same degree, and when it is deficient the loss must be compensated by a greater amount of mental concentration than is necessary in those in whom the imitative faculty is prominent.

But increased mental concentration is also still more necessary in those who, although able to imitate mechanism of sound production which they see, are nevertheless unable to reproduce sounds which they only hear. And this is the case with the present patient. He can only produce certain sounds so long as his visual attention—which soon wanders—can be commanded.

It may be asked how can his visual and auditory memory be deficient considering that he has committed to memory a large stock of rhymes, short poems, and hymns, which he repeats glibly in his own tongue?

It is generally true that a good memory is either auditory or visual. Those who possess a good auditory memory seem to hear the words which they have memorialised dictated to them, whilst those of good visual memory are able to visualise the printed page, and literally read it aloud.

Most learned anatomists and also artists possess the faculty of visual memory to a marked extent, whilst the auditory memory is perhaps more commonly met in uneducated people. The accuracy with which the latter will often repeat the exact words of a long conversation, which could only be recalled in substance by those of higher social position, is astonishing. Doubtless the visual memory in educated folk has increased at the expense of the auditory, by the artificial aid of writing and reading.

Those who neither possess a good auditory nor a good visual memory are compelled to rely on the association of ideas for their power of recollection, and this will be more or less accurate as the association is logical or arbitrary.

My patient understands everything that is said to him. Words, whether spoken in his own tongue or correctly pronounced, convey a definite meaning to his mind which he remembers, although he

seems not to remember the exact sound which he hears, or the method of producing it.

He appears to act on the principle advocated by that whimsical philosopher the late Lewis Carroll,—“Take care of the sense, and the sounds will take care of themselves.”

Neither his eye nor his ear aids him. He remembers the meaning of words, but not the sounds of the words themselves, nor the mechanism of their production, nor their appearance in print, as he cannot read.

Comparison with other cases.—For the purpose of comparison, the following table, showing letters of the alphabet as pronounced by six cases, is appended.

LETTERS OF ALPHABET AS PRONOUNCED IN SIX CASES OF IDIOGLOSSIA.

A	{	a (Guthrie) ah (Guthrie) ah (Hale-White, Golding-Bird) ahsee (") ādē (Fred. Taylor) ah (Hadden)	B	{	b b b b bēder be
C	{	tsee t or d c c dēder ve	D	{	d t or d d gee dēder te
F	{	ap or ep ā ā ahth F arder fish	G	{	tee d yee dze dgeeder te
I	{	ai ah ah aee (a as aye) aider ah	J	{	teh dā (as in day) eeyah zaee (a as aye) diader vah
L	{	al a a ahl L aider ve	M	{	em em or ahi ahm em em ve
O	{	o o ū (as in fru- gality) o ōēr vah	P	{	p p p p p bēder pe
R	{	ah ah ah ah arder ah	S	{	ats ā ā ahsee ees aidow fish
T	{	t t t kee deder te	Q	{	too koo t ū q u ve
H	{	aitch ah ahsee aisee (a as aye, i as aye) aidow vatch	K	{	tā K (normal) tah kee dader vah
N	{	ain air ahdi n en ve	V	{	v v v v v v v

U	u ōō or dōa u (as in few) u (as in fru- gality) ūer ve (ʔ)	V	wee b yee wee veder ve	W	dubbleyew dādo dahbeyew dahbeeyew doubbeouer dedorch
X	ats ak aht ēs aidow fitch	Y	Y bāā eeyah y whyder vah	Z	dads dair yahddī z diader ve

It will be seen that they all bear a general resemblance to each other, but the individual peculiarities are so many that it would be tedious to analyse the list in detail.

The general resemblance consists in the fact that sounds of which the mechanism of production is obvious, presented the least difficulty, whilst the difficulty in most cases increased in accordance with the obscurity which attends the visible method of formation of each individual sound. Dr. W. S. Colman (op. cit., p. 1421) sums up the characteristics as follows:

"Letters which cannot be pronounced include in almost all cases the posterior linguo-palatals, the labials and linguo-dentals nearly always escaping. Next, the gutturals F, V, R appear to give most difficulty. The average number of consonants which patients failed to pronounce was eight, and for these nearly all substituted T or D, the former for voiceless and the latter for voiced consonants. In some cases it was easier to pronounce consonants when they were terminal than when they were initial. . . . In all cases pronunciation of vowels has been good, and there has been an attempt at substitution."

In the main this summary holds good. The explosives—B, P, for instance;—the labial M, and the linguo-dentals T, D, were more frequently heard than the sounds G, H, J, K, F, V, R, C (soft), X, S, and Z, the mechanical production of which is obscure, but the same sounds were not substituted by all alike for those most difficult of production.

In all cases consonant sounds were substituted for each other, but the consonants were not in all cases the same. In several the curious fact is noticed that elementary sounds appeared in combination, but not alone. Sometimes they were pronounced when terminals but not when at the beginning of a word, sometimes the reverse. The cases which show the greatest individual peculi-

arities are those of Dr. Hadden and Dr. Frederick Taylor.

In Dr. Hadden's case the sound V (ve) stood for the consonants C, L, M, N, Q, Z. It also appeared in H (vatch), K, O, and Y (vah). F and S appeared as the word "fish," and X as "fitch." This peculiarity may have been traceable to the habit of biting the lower lip, common in children. This would lead to the production of F or V sounds amongst those first articulated. Generally, but not invariably, labials and linguo-dentals are first heard—in children learning to talk. In rare cases gutturals are earliest.

In Dr. Frederick Taylor's case, in addition to mispronunciation and substitution of consonants for each other, there was the addition of a suffix "aider" or "er" to each letter or word. It was noticed when he first began to talk at the age of two. After careful instruction had been given, Dr. Taylor says, "It was chiefly when he repeated a string of words or a phrase after me that he became unintelligible, and the apparently involuntary affix 'eeda' or 'ida' was produced."

Somewhat similar suffixes are common enough in those who drawl, whether from affectation or from a paucity of words. There is always, however, a pause between the word and the suffix, which prevents their speech from being unintelligible, as it would become were they to speak fast, and add the suffix without the intervening pause.

In this connection Dr. Taylor alludes to the schoolboys' game in which a meaningless syllable is added to each word. Dr. Wyllie ('Disorders of Speech,' p. 130), quoting Kussmaul, says that amongst German children the practice is known as the "Erbsen speech game," from the addition of the syllable "erbsen" to each monosyllabic word.

It is possible that in Dr. Taylor's case the suffixes arose from an attempt to emphasise final consonants.

In singing, the necessity of prolonging the final sound of a consonant ending a word is sometimes inevitable; for instance, "and" and "yet" become "ahnd-der" and "yet-ter," thus affording some parallel to the employment of the suffixes "ida," "eda," by Dr. Taylor's patient.

Finally, the general effect produced on listening to the speech of these children is that of hearing

an unknown tongue, until one is initiated into the mysteries which underlie it. It then is discovered to be merely the Queen's English, the words of which are mangled and distorted, but English all the same. The language flows easily and without hesitation from their lips. It is polysyllabic, and in my own case, at all events, each syllable can be clearly uttered. In most, however, there is a tendency to slur over words, which renders their sounds difficult to imitate. One of Dr. Hadden's patients was unable to understand his own language when spoken by others, doubtless because it was difficult to imitate. My patient understands the meaning of words whether pronounced correctly or as he does. He calls a lion "dai," and a sheep "teeër," but if asked to find one of these animals by either designation in a picture-book he points it out at once.

The vocabulary is extensive; inflections are indicated, and grammatical construction is correct in form. Every word in a sentence is represented by a separate sound. In these respects it differs from "baby English," which it resembles, however, in the substitution of consonants for each other. Gestures are not used except when the child finds it impossible to make itself understood, and even then the pantomime is feeble.

Use of term idioglossia.—Objection may be raised on the ground that this is not a peculiar language, but, as already stated, imperfectly pronounced English. Yet practically the result is the same as if a new language were heard, and so the term may pass. It is not found in Greek classics, but it is quite legitimate to coin it from the word *ἰδιόγλωσσοι* (of distinct, peculiar tongue), found in Strabo.

Professor Wyllie prefers to use the word "lalling" (German, *lallen*, "to speak as a child") to include idioglossia amongst all imperfect performances of the oral articulative mechanism. But, as already mentioned, idioglossia is not simply "baby English."

One may altogether exclude the notion that idioglossia is a species of "atavism" or a "sport in language," by a study of which we may obtain a glimpse of the early evolution of speech as it occurred amongst our prehistoric ancestors.

This possibility has been dear to philologists since the days of Psammitichus, who, Herodotus tells us (Book II, ch. ii) caused two new-born

infants to be isolated for two years and suckled by goats. At the end of this time the only word the children could say was "bekos," the word "bek" being Phrygian for "bread." Hence Psammitichus concluded that Phrygian was the most ancient language in existence. The Scholiast, however, takes the commonplace view that the children were merely trying to imitate the bleating of the goats, which is probably correct.

In more recent times other attempts have been made to trace in childish utterances the germs of language, and to show that children in using "words of their own invention" hark back to the speech of their ancestors. Thus Horatio Hall ('Proceedings of the American Association for the Advancement of Science,' vol. xxxv, 1886) gives an elaborate account of twin children of German extraction who spoke a language of their own, in which their family detected a German accent, although the German language was never spoken in the household. Similarly, Dr. E. R. Hun ('Monthly Journal of Physiological Medicine,' 1868) records the cases of a girl aged 4½ years, and her younger brother, who invented a language, some of the words and phrases of which had a resemblance to French, although it seemed unlikely that the children had ever heard French spoken. Whether this was so or not, the resemblance to French in the instances given does not seem particularly striking. Nor do the examples cited of the other children's diction suggest a German origin. Romanes ('Origin of Human Faculty,' p. 141) says in reference to Hun's cases, "Amongst the words showing this resemblance are *feu* (pronounced, we are expressly told, like the French word), used to signify fire, light, cigar, sun; *too* (the French *tout*), meaning all, everything; and *ne pa* (whether pronounced as in French or otherwise we are not told), signifying not. *Petee petee*, the name given to the boy by his sister, is apparently the French *petit* (little), and *ma* (I) may be from the French *moi* (me)."

Of these it may be said that *feu* is just as likely to have been an attempt to say *fire* as its French equivalent. *Too*, signifying all, everything, is as much English as French. *Petee petee* is as likely to be an attempt to say *pretty, pretty* (an expression commonly used in talking to babies), as the French word *petit*. *Ma* might equally well stand for me as *moi*.

Ne pa for "not" is more difficult to explain. But *ne* or *neh* is commonly used for "no" by children, and we are told that this child could say *papa*, *mamma*. Perhaps its father was less indulgent to it than its mother, and so it associated negation with him. Such an association of ideas is not more grotesque than others which account for the supposed use of "words of own invention" by children. *Do children ever really invent language?* and are "*words of own invention*" used by the subjects of *idioglossia*? Children often—perhaps always—when learning to talk appear to use words of their own invention. Yet I believe that for the most part their words are but imperfect attempts to imitate those which they hear. After the period of "*echolalia*," as it is called—in which sounds are imitated parrot-like—has passed, children begin to attach a definite meaning or connotation to the words they use. Frequently the connotation of the name of an object, or its attributes, is used as the name of the object itself, and of all other objects between which the child detects a fancied resemblance. The connotation may exist only for the child, and unless one knows what it is, the child will appear to have coined a new word, especially when the word which it wishes to utter is imperfectly pronounced. Thus a little boy aged two years called grapes *bazoons*, and on first seeing a sign-post asked whether it was not a *zebra*. He had recently been playing with a toy balloon, and the sign-post happened to be covered with striped advertisements. Hence the association of ideas based on superficial resemblance. His name for umbrella was *bessersole*, evidently a combination of umbrella and parasol.

A good example of the use of attributes for the names of objects is given by Taine ('*De l'Intelligence*,' vol. i, p. 48). A child aged eighteen months was told of everything hot, "*ça brule*." After playing "*cou-cou*" with its mother, it saw the red sun sink behind a hill, and said "*a bule cou-cou*," i. e. "*the sun is playing hide and seek*."

It will often be found that the supposed new terms are but misapplied and mispronounced words which children have actually heard and imperfectly understood.

Thus in the instances given by A. Crane in '*Nature*,' April 9th, 1891, p. 534, a child is said to have called an ordinary chair *lakail*, a great

arm-chair *lukull*, whilst a little doll's chair was *likill*.

Obviously the word *likill* was the child's version of *little*. The doll's chair would inevitably have been introduced to the child's notice as "*a little chair*," and the attribute *little* or *likill* would be regarded as the name of the chair itself, whilst in trying to discriminate between this and other chairs various vowel sounds were interpolated.

It is not surprising that the term *little* should have had such wide significance for the child, because in the nursery it is accustomed to hear every portion of its person, its clothing, and all that concerns it referred to as "*little*."

The second instance given in support of the view that children invent language is that the root for all round objects was "*m m*"—a watch, plate, and the moon were *mem*, a large round disc or table was *mum*, whilst the stars were *mim*, *mim*, *mim*. Here, again, the explanation is obvious. "*M*" is one of the earliest sounds which a child learns to articulate, and its attention is sure to be drawn to the full moon on occasions when its male parent endeavours to soothe it during the silent watches of the night. Thus it soon learns to associate the recently acquired "*m*" sound with the moon, and therefore with all round objects. The various vowel sounds, which are also soon learnt, were doubtless used in order to distinguish differences in magnitude of circles. The repetition of *mim*, *mim*, *mim*, for "*stars*" obviously was an attempt to express the idea of plurality.

The origin of some of the words drawn from the vocabulary of children to whom Romanes has alluded is obscure, but had not the clue been lost it is probable that, however meaningless or arbitrary the words used may have seemed, they admitted of explanation.

They may have been derived—

(1) Simply from imperfect articulation of the real names of objects.

(2) From imperfect articulation of attributes, real or imaginary, of objects used as the names of the objects themselves.

The origin of some may have been purely onomatopoeic—e. g. *migno migno*, signifying "*water*;" *ni si boa*, "*carriage*," &c.

Hence it is open to doubt whether children ever actually invent language, rather than distort such language as they hear. The strongest argument

against spontaneous evolution of speech is the fact that deaf-mutes never talk at all unless taught to do so. Idioglossia is not baby language, neither can it be strictly regarded as a new tongue, although many of the words used seem absolutely unlike those which the child has heard. The words *dâi* and *pâi* used by my patient do not in the least resemble *lion* and *fly*, yet they are not invented names for these creatures, but are the child's own version of their names as heard by him. I do not believe that children ever, except perhaps in play, *invent* new words, and am convinced that idioglossia is not an instance in favour of a contrary supposition.

NOTES, ETC.

The Local Treatment of Acute Articular Rheumatism.—'La Presse Médicale' of February 5th, 1898, gives the following treatment for this condition:—The local use of an ointment composed of liquid vaseline 1 oz., salicylate of methyl $\frac{1}{2}$ oz.; this is smeared upon lint and bound about the joint, and the lint is now in turn covered with gutta-percha. In other cases an ointment composed of salicylic acid, as follows, may be used:

R Vaseline... .. 1 oz.
Salicylic acid 45 grs.

In still other instances salicylic acid can be gotten into the system through the skin by the following means:

R Salicylic acid 1 dr.
Salicylate of sodium ... 60 grs.
Extract of belladonna... 15 „
Vaseline 1 oz.

This is applied in the same way as the ointment of the salicylate of methyl.

Salol is said to be especially useful in gonorrhœal rheumatism. It may be dissolved in ether and used in the following prescription:

R Salol 1 dr.
Menthol 30 grs.
Ether 1 dr.
Lanolin... .. 1 oz.

Guaiacol, when locally applied, is also a powerful pain-reliever as well as an antipyretic. It may be painted upon the part in the following solution:

R Pure guaiacol 1 dr.
Alcohol (85 per cent.)... 1 oz.

One fourth of this mixture is sufficient for one application. Too frequent application of it will cause too great a decrease in temperature. After it is applied the part should be covered by an air-tight bandage. The following ointment may be used in its place:

R Guaiacol 1 dr.
Vaseline 1 „

Or the following used:

R Guaiacol 1 dr.
Salicylic acid 30 grs.
Salicylate of methyl ... 1 dr.
Vaseline 1 oz.

This mixture is very active, but possesses an intensely strong odour.

Sometimes good results follow the application of the following prescription:

R Guaiacol 1 dr.
Terpinol 3 drs.
Alcohol (85 per cent.)... 4 „

Therapeutic Gazette.

A Hitherto Unknown Secondary Effect of Trional.—Kaempffer reports a peculiar effect of trional in four cases of carcinoma, the drug producing a wakefulness such as is occasionally seen as the effect of opium and some of the alkaloids. His patients suffered some weeks before the fatal termination of the disease from sleeplessness, and he gave each 15 grains of trional, with the result that instead of getting an hypnotic effect, a peculiar state of excitement was developed, which lasted through the night. The patients, no one of whom was addicted to alcohol, and who had all taken the remedy in the prescribed manner, stated that they had noticed a certain fatigue soon after taking the medicine, but had not been able to go to sleep. Soon after this they were seized with extreme restlessness associated with fear and heart palpitation, which grew worse and worse, until finally a delirium set in. Two of them even had visual hallucinations, and during the attack of fear had jumped out of bed. This condition had continued till toward morning, followed by several hours of a restless, superficial sleep. In one of the cases a somewhat sounder sleep, lasting six or seven hours, had resulted when the dose was reduced to $7\frac{1}{2}$ grains, and it had been administered late in the

afternoon ; in the other three cases, however, there was no difference in the results, nor was there upon increasing the dose to 30 grains. In this latter instance the condition of excitement was still further increased, so that the administration of trional had to be discontinued. — *Therapeutische Monatshefte*—*Occidental Med. Times*.

Etiology and Operative Treatment of Tubal Gestation.—Dührssen describes twenty ventral and fifteen vaginal coeliotomies, two cases of posterior colpotomy, and three vaginal total castrations for tubal gestation, and gives the results ('Archiv für Gynäkologie,' Bd. liv, H. 2). In those cases alone in which the tumour reaches some distance above the pelvic brim does Dührssen advise ventral coeliotomy. The advantage of vaginal coeliotomy is the comparatively small operation as opposed to a laparotomy; cure, healing of the wound, and recovery are more rapid by this method; shock is less: abdominal incision through a fat abdominal wall often causes ventral hernia, which hernia may later lead to serious complications. He describes his vaginal technique. He advises the drawing out of the tumour with broad or pointed clamps, since catgut ligatures may cut through or tear off the tumour. He, further, rarely does a vaginal fixation after the extirpation; he says that the abdomen or vagina should be entirely sewed up, in order to prevent adhesions between uterus and vagina, which cause complications in later pregnancies and labours. He advises extirpation of the tube in tubal gestation, even when no hæmorrhage exists, and when pain alone is present; for even after the death of the foetus dangerous hæmorrhages may occur. He speaks against the killing of the foetus by morphine injections. Etiologically, Dührssen considers tubal disease, particularly gonorrhœal infection, as the most frequent cause of extra-uterine gestation.

Medical Record, July 23rd, 1898.

REVIEW.

Manual of Operative Surgery.—By H. J. Waring, M.S., M.B., B.Sc.(Lond.), F.R.C.S., Demonstrator of Operative Surgery and Surgical Registrar, late Senior Demonstrator of Anatomy, St. Bartholomew's Hospital, Surgeon to the Metro-

politan Hospital, &c. (Young J. Pentland, Edinburgh and London.)

So much has been written on operative surgery that one naturally turns with some curiosity to the préface of this work by Mr. Waring, and finds that this book has been written with the object of serving as a text-book for the classes held in the Operative Surgery Department of St. Bartholomew's Hospital. The author has succeeded in less than 700 pages in the difficult task of considering and describing the various surgical operations which may be performed upon the human organism for the alleviation or radical cure of those malformations, diseases, or injuries amenable to direct surgical interference. The subject is divided into twenty-eight chapters, and there are 428 illustrations scattered through the book. The care and labour which has evidently been lavished upon this production is well shown in the fact that most of the illustrations have been specially prepared. A very useful feature, which should prove of great advantage, is the addition of a short account of the main indications for the performance of an operation or operations immediately before the description of such operation or group of allied operations. Each operation is discussed under six headings:—(a) The indications for its performance. (b) Preliminary preparations—(1) for asepsis; (2) for anæsthesia. (c) Selection of instruments. (d) Position of patient, operator, and assistant or assistants. (e) Actual operation. (f) After-treatment. In consequence of this most excellent arrangement, and aided by a good index, the contents of this valuable work of Mr. Waring's can be consulted and referred to with great ease and no waste of time. As we have little doubt but that a second edition of this publication will soon be required, we hope that the author will still further increase the importance of this remarkable production by devoting if possible a little more space to the consideration of the operations for removal of the Gasserian ganglion, to operative treatment of fractures of the patella, and to operations upon the spinal column and the spinal cord. This book, possessing the advantage of not only being well written but also of being well printed, is sure of a good reception from a far wider public than the students attending the classes held in the Operative Surgery Department of St. Bartholomew's Hospital.

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IN THE WARDS OF CHARING CROSS HOSPITAL WITH DR. ABERCROMBIE,

July 7th, 1898.

Hypertrophic Cirrhosis of the Liver.

THIS patient is 71 years of age, and only came in yesterday. As you see, he has got a high degree of jaundice, and we are told that it came on three weeks ago, when he noticed he began to get yellow, and was feeling unwell. He consulted a doctor, who told him he had got jaundice, and advised him to come to the hospital. He gives no history of pain or vomiting, or gastric symptoms of any kind; he complains of nothing but increasing weakness and jaundice. When I saw him in the ward for the first time yesterday, before asking him any questions, I quite expected to find some form of malignant disease, having regard to his age and general appearance. One position at which malignant disease is likely to give rise to jaundice, particularly in elderly people, is at the head of the pancreas. However, when I came to examine him and to hear his history, I was bound to admit that it did not seem very probable. For the disease to be malignant we ought to have had a longer history of ailment, and if he had pancreatic disease he ought to have been wasting, and he would also have had some gastric symptoms, if not actual pyloric obstruction. On examination, however, all we make out is a very decided enlargement of the liver, and especially of the left lobe, which is smooth, and the surface feels firm. It is not tender, and the spleen does not appear to be enlarged. With these signs, and in the absence of ascites, we may regard atrophic cirrhosis of the liver as excluded. You do not get jaundice in the ordinary alcoholic cirrhosis until late in the disease; and ascites would be sure to precede by a long way the occurrence of jaundice. His spleen is not enlarged, and that is against the idea of ordinary cirrhosis, because

then the portal system would be obstructed, and the spleen always more or less enlarged. One very important point is that his motions contain bile; that is really the crucial thing in the diagnosis, and leaves many things out of court—pressure on the duct, either internal or external, gall-stones, &c. There cannot be obstructive jaundice when there is bile in the motions. He has passed two or three motions since he has been in, and they have all contained a good deal of colouring matter. Secondary carcinoma of the liver might be associated with a non-obstructive jaundice, but his liver enlargement is not due to that because the surface of the liver is perfectly smooth, and you know that in secondary carcinoma of the liver there are distinct nodules of varying sizes on the surface which can be easily felt. Moreover, his history does not suggest malignant disease. Lardaceous liver can be excluded at once; he has not any source for lardaceous disease as far as we know. The only disease which I consider fits all his signs and symptoms is that somewhat rare form called hypertrophic cirrhosis, or biliary cirrhosis. Against this is the fact that the liver is not so big as usual in that disease. In hypertrophic cirrhosis you generally have a very large liver, quite smooth, no ascites, no enlargement of the spleen, marked jaundice, generally fever or febrile attacks, and very often some mental symptoms; there is a tendency to stupor or a subdelirious condition. These symptoms our patient has; you will notice he is now talking in a rambling manner. Therefore one is practically driven to the diagnosis of biliary cirrhosis. Of course we have it in a comparatively early stage, and the liver may enlarge a good deal more yet. He tells us that he has always lived in England. His occupation has been that of commercial traveller for a firm of jewellers, a calling frequently associated with habits of intemperance, and hypertrophic cirrhosis of the liver seems to have a close association with alcohol.

In the matter of treatment there is not much to say. I am inclined to go in for mild mercurial purges from time to time. I believe biliary cirrhosis is a more progressive disease even than atrophic cirrhosis. What the mental condition is due to it is difficult to say, but you may often see people more deeply jaundiced than this patient, and there may be no mental symptoms whatever.

Aneurysm of the Aorta.

This man is a clerk aged 42, but he has not always been a clerk; he was at one time on omnibus work. He comes complaining of pain in his chest. There is a history of his having had rheumatic fever two or three times, but the essential part of his history is that four months ago he began to suffer from pain in the præcordial region, and the pain went down the left arm, and was increased on exertion. The attacks of pain became very frequent, and led to his feeling faint, and resulted in his taking medical advice.

The first thing discovered about him was that he had a well-marked diastolic murmur at the base of the heart, and the question arises whether he has aortic regurgitation, or whether, in view of his history, he has not got an aneurysm of the arch of the aorta. I am inclined to think if he has not got an aneurysm he has at any rate a dilatation of his aorta, especially that portion of it where the innominate artery is given off. He has one small patch of dulness at the base beneath the sternum, but no definite pulsation; there is no supra-sternal pulsation, and no tracheal tugging. But he has one sign which cannot be overlooked, namely, very marked pulsation in the vessels at the root of the neck on the right side; as compared with the other side the difference is very distinct. Whether there is a difference in quality of the pulsation at the wrists is fairly open to dispute. I am inclined to think that the right pulse is very slightly weaker than the left. There is no inequality of pupils. The patient has described the pains very well; that is to say he says they shoot up to the axilla and then down the arm, and the route which he describes corresponds to the course of the musculocutaneous nerve. He could not know that there was a nerve there, and therefore I think his statement must be accepted, and it constitutes very valuable evidence indeed. I infer from the difference between his radial arteries that the dilatation involves the innominate to some extent, but not enough to give rise to pulsation in his thorax. He has got one other sign, namely, that he is a little hoarse, but the value of it in this particular case we are unable to determine. Hoarseness is something which you look for in a case of suspected aneurysm. Possibly he has some paralysis of his left recurrent laryngeal nerve by pressure on

that nerve as it turns round the arch of the aorta ; but unfortunately he has some ankylosis of his jaw, which prevents him opening his mouth more than three-quarters of an inch, and therefore a proper examination of his larynx is not possible. It is fourteen years since he had a blow on the jaw which caused the ankylosis.

As regards the aetiology I assume that all aneurysms are syphilitic. Aneurysm is always due to disease of the vessel, and I believe that disease is practically always syphilis.

I had in the hospital two or three years ago, a case possibly of traumatic origin, because it was associated with exertion on the part of the patient who was a police officer. He had a severe tussle with burglars, and felt a pain in his chest shortly afterwards. I do not know how long afterwards he presented himself at the hospital, but he had very distinct signs of aneurysm. Under treatment he did extremely well, and that aneurysm is practically consolidated ; there is no longer any pulsation, and he has no symptoms. I remember another case many years ago of a young man who had an aneurysm of undoubtedly traumatic origin. He was swinging a heavy weight round his head a number of times for a wager, when he felt a very severe pain in his chest. He came to the hospital within a week, and he already had a pulsating swelling in the chest, and developed a very large aneurysm, which under treatment became completely consolidated. I believe aneurysms that own a traumatic cause, or in which traumatism plays a part, are the most favourable for treatment. I do not propose to put our present patient on a strict Tufnell diet. I shall restrict his fluids, and give him large doses of iodide of potassium. Very few patients can stand the strict Tufnell treatment, which should not be commenced unless there is reasonable probability that the patient will be able and willing to endure it.

Acute Tuberculosis.

This little girl is 2 years old. She comes in with a history of cough, and with a fluctuating temperature. She had been ill a fortnight when she came in, and has now been in ten days. When I examined her on admission she had extremely well marked signs over the right chest of pneumonia, or rather broncho-pneumonia. There were some râles at the anterior base, and râles in the

axilla, and all over the back, with intense bronchial breathing. There was also a small patch of well-marked bronchial breathing on the right side at the root. I was inclined to regard the case as one of broncho-pneumonia, which I thought was going to spread to the other side. But the latter part of my expectation has not come about, as the signs have not extended to the other side. You will notice that on the left side she still has very abundant crepitations over the back and in the axilla.

In all these cases the question you have to solve is, is this broncho-pneumonia or is it tuberculosis ? To my mind it is a very difficult question, and the first time you see the patient it is a question which I think you had better leave unsolved. Now when you get broncho-pneumonia which does not speedily clear up, you must always think of acute tuberculosis. The thing that I look for especially in these cases is enlargement of the spleen. In cases of acute miliary tuberculosis the spleen is always involved, and it is generally involved early, and then it swells up and you can feel it. If, therefore, with catarrhal signs in the lungs you have enlargement of the spleen in a young child, the chances in favour of tubercle are immense. Of course it might be possible that the spleen is enlarged from congenital syphilis, or the patient might be the subject of leucocythæmia, and of course those are matters which you would have to inquire into and exclude. Otherwise, the enlargement of the spleen with the other signs practically means tuberculosis. A child was quite recently in this ward with general pulmonary catarrh, enlarged spleen, and fever, and in confirmation of the diagnosis of acute tuberculosis there was a very interesting physical sign, namely, that close to the sternum at the second left rib and interspace we got a patch of intense bronchial breathing ; it was surmised that the bronchial and tracheal glands were very much enlarged and caseous, and that this would account for the conduction of the sound of the bronchial breathing to the ear, and at the post-mortem examination this surmise was completely verified.

To-day for the first time I have been able to feel this little child's spleen ; I have hitherto held out hopes that the case might be one of simple broncho-pneumonia, but I do not now hesitate to say that the child is tuberculous. She has had, at some

time or another, a suppurating gland in her neck. I do not believe every enlarged gland of a child is tubercular, but when you get other signs of tuberculosis you are practically forced to go back and admit that the gland was tubercular too. Probably in a week or ten days this child's spleen will be very much larger. Again I would say that I regard that as a very valuable diagnostic sign. I do not think it is of much use to percuss a spleen. I admit that a spleen may be enlarged to twice its normal size without your being able to feel it, but I like to be able to feel it before I say definitely that it is enlarged. In these cases I believe the spleen is often the first organ to be affected. In post-mortem examination you find the tubercles in the spleen more abundant than anywhere else; they are also larger and yellower.

Tubercular Peritonitis.

Our last case is another little child, the subject of a more chronic tuberculosis, that is to say, abdominal. These two patients illustrate the two common varieties of tuberculosis in children, the acute miliary and the abdominal. Here is this patient's chart; you will see that though there is not much fever, there are great variations. These variations of temperature have almost as much meaning when they occur with subnormal temperatures as when they occur above the normal. This child no doubt has chronic tubercular peritonitis. In the abdomen, just below and a little to the left of the umbilicus, you can feel a little ovoid very hard mass as large as a cherry stone. No doubt it is a mesenteric gland, and there are masses of induration to be felt on both sides of the abdomen. Since she came in, the abdomen has become much more distended and more doughy, no doubt from distension of the intestines with flatus in front of the induration. You will notice how languid and listless the child is; it is the usual state of the subjects of tubercular peritonitis. I have never seen her smile, or heard her cry.

Up to a certain point some of these cases are very satisfactory to treat, but this one has been extremely resistant; she has not shown any sign of improvement whatever. We have used mercurial inunction, in which I am a great believer in all forms of peritonitis, the ordinary unguentum hydrarg. twice a day, either rubbed in or bandaged

on. We are now trying to give her some iodoform in treacle, which she appears to be taking fairly well. In her case there is probably intestinal ulceration too. I think in cases where there is no ascites indurations are often secondary to tubercular ulceration of the bowel, which need not be attended by diarrhoea; constipation is quite as common.

I am inclined to advise that later on the abdomen should be opened and some iodoform put in. The chief objection to such a procedure is that in these cases of ulceration the bowel gets so brittle that in trying to separate the adhesions you might tear the bowel, but nevertheless I think it is fair to try whether anything can be done for her by abdominal surgery. I do not propose to do more than simply open the abdomen and put some iodoform in, and probably flush it out with some mercurial solution.

When there is ascites the prognosis is not nearly so bad. I do not know why; it is the same disease, only in a different form. I have had many cases of ascites of tubercular origin which have done very well. I have a young man in the hospital now who has lately come in for bronchopneumonia. Some three years ago he had a large serous effusion into the left pleura, which was accompanied by a good deal of fever, always creating a suspicion of tuberculosis. Six months afterwards he came back with well-marked ascites, for which we could find no other explanation than that of tubercular peritonitis. He made a good recovery, and tells us that he has been quite well since and able to work hard until his present illness, which began a few days ago. There are no signs whatever in the abdomen now, so that his recovery has been quite complete.

Relationship of Migraine to Epilepsy.—

Rachford ('Amer. Journ. of the Medical Sciences,' April, 1898), from observation and study of the literature, is convinced that the kinship of migraine and epilepsy is one of the facts and not one of the theories of medicine. He believes that he has proven that paraxanthin is a most important factor in true migraine, and that it is also an important factor in the production of one form of toxic epilepsy. He says migraine therefore bears a close ætiological relationship only to toxic epilepsy.—*University Med. Mag.*, July, 1898.

INCIPIENT PULMONARY TUBERCULOSIS.

A Lecture delivered at the Hospital for Consumption and Diseases of the Chest, Brompton, June 8th, 1898,

By F. J. WETHERED, M.D.Lond., F.R.C.P.,

Assistant Physician to the Hospital.

GENTLEMEN,—I have no doubt that in dealing with the subject of incipient pulmonary tuberculosis you would rather that I should lay stress on the diagnosis, prognosis, and treatment rather than enter into a deep scientific discussion as regards the nature of pulmonary tuberculosis only ; so that although I think it advisable in a subject like this to take the ordinary text-book divisions—ætiology, pathology, symptoms, and treatment, I shall have very little to say about ætiology and pathology.

By pulmonary tuberculosis we mean a disease of the lung which has for one of its causal factors the *Bacillus tuberculosis*. Because a patient gets his lungs infected with tubercle bacilli it does not necessarily follow that the same series of symptoms will be produced. We have the ordinary form of pulmonary tuberculosis, which is perhaps best known as fibro-caseous, in which there are changes going on in the lungs, first of all of a caseous kind, and then nature makes an attempt to heal the disease, and we have a fibrous process established. That is by far the most common variety. Then we have what is known as "caseous pneumonia," in which the whole of one lung becomes consolidated into a caseous mass. Next there is the fibroid, in which from the beginning the disease takes on a fibroid type. We have also various other types, such as are described by Sir R. Douglas-Powell, namely, "florid phthisis," and what he describes as "pulmonary tuberculation." I have on previous occasions considered more minutely the differences between these forms, so that I need do no more than mention them. I shall deal almost exclusively with the fibro-caseous type to-day.

Regarding ætiology, it is of course essential that we should have the tubercle bacillus, but, fortunately for mankind, the tubercle bacillus itself is not sufficient to cause the disease ; we must have something in addition to the tubercle bacillus.

What that something is we do not precisely know. Some observers call it "deficient resistance," others call it "unsuitable pabulum," and various terms of that sort are employed. If the tubercle bacillus alone were sufficient to cause the disease, probably every one who is connected with this hospital would be affected with consumption. That gives rise to the question, can the tubercle bacillus attack a healthy lung? In the very large majority of cases, probably, the tubercle bacillus cannot attack a healthy lung. The only way in which a healthy lung may be affected is where the dose of the poison is very great—that is to say, if for some reason a person inhales very large doses of the poison, it is possible for him to take the disease ; but otherwise I think we may take it that the tubercle bacillus cannot attack a healthy lung.

The idea has become prevalent that the tubercle bacillus is omnipresent, that is to say, is found everywhere. It, however, seems to be extremely doubtful whether the tubercle bacillus is so widely disseminated as was at one time thought. When I was pathologist at the City of London Hospital, Victoria Park, I tried very hard to catch tubercle bacilli in my laboratory. There I had sputum to examine daily. I also dried sputum in open plates and then tried to find tubercle bacilli in the dust, and in other parts of the room, but in that I failed. Since that time other experiments have been tried with the same results. Dust from various parts of the hospital was taken by Dr. Heron and Dr. Chaplin, and inoculated into guinea-pigs, but (if my memory serves me correctly) only in two cases at most did those guinea-pigs get tuberculosis.

Every room in this hospital opens into a ventilator, and these ventilators gradually converge until they enter two extraction shafts. At the top of these is a coil of steam pipes, the heat of which causes a draught sufficient to blow one's hat off. I remember some years ago, whilst I was house physician here, Mr. Taylor (who was then assistant resident medical officer) and myself tried some experiments. Over the opening we placed a large funnel, and in the end of the funnel we placed cotton wool, so that the air which came out through it had to pass through that cotton wool. After a number of days we dissolved the wool in collodion and examined the deposit, but we utterly failed to discover tubercle bacilli. Therefore it is

very doubtful, especially in places like this hospital, where every precaution is taken against dissemination of the tubercle bacilli, whether the bacillus is as widely distributed as was thought.

At one time heredity was one of the essential points as regards the ætiology of pulmonary tuberculosis, but now-a-days medical men are beginning to discredit the part which heredity plays in pulmonary tuberculosis. The discovery of the bacillus was almost a death-blow to the theory of heredity, which at one time held such a high place. Of course I will not say it has no influence, but it very rarely occurs that the disease is transmitted direct from the parent to the child. It is probable that a tendency to general weakness of the whole system is transmitted from parent to child, and that weakness may account for the individual finding some difficulty in resisting the disease; but if that tendency is recognised and every precaution is taken against it in early life with respect to surroundings, &c., there is no reason why the offspring of delicate parents should suffer from pulmonary tuberculosis. There are one or two matters which I will just briefly refer to.

First of all as to insanitary conditions. It has been proved over and over again that one of the chief elements which have to do with the transmission of pulmonary tuberculosis is moisture, and in very recent reports I have seen that towns which at one time were badly drained had a very high death-rate from consumption, but as soon as the drainage was properly seen to the death-rate from consumption fell very considerably.

Then, of course, all debilitating circumstances tend more or less to lessen the resistance-power of the organism. Frequent child-bearing is a very marked predisposing cause indeed; so also is prolonged lactation. The prognosis of pulmonary tuberculosis in married women is a great deal more serious than it is in the unmarried. I need not say anything about occupations. As regards age, the results are fairly well known—namely, that the older the age at which the disease is contracted the better is the prognosis. If a young adult of between fifteen and twenty develops the disease, the prognosis is worse than if a person does not contract the disease until thirty-five or forty. Previous diseases have some effect, such as pleurisy, and I am at present engaged in taking out the statistics of several thousand cases of patients who

have been in this hospital with regard to previous history, age, and so on, which I hope will eventually prove of some assistance, as far as statistics can prove of assistance.

I shall not now go into the pathology, but will pass on to consider what are the symptoms of incipient pulmonary tuberculosis, and in that I shall refer entirely to the fibro-caseous type, where the onset is insidious, and not to the acute varieties such as miliary tuberculosis and caseous pneumonia, I shall consider only the cases we see mostly here, and which constitute about 95 per cent. of the total.

Our first difficulty in diagnosis is that of obtaining a history, and that depends very much upon the class of patient. Of the out-patients at this hospital nearly all of them, if you ask how long they have been ill, date the commencement from the time they were obliged to give up work; but on cross-examining it will be found that they were ill for a considerable period before that. We shall probably find that they say they have been gradually ailing, that they have not felt quite so well as usual, that there has been gradual failure of strength, and they will perhaps tell you that their friends have been noticing of late how pale they have been getting. After this failure of strength, anæmia, and cough develop. This cough may be only very slight at first, perhaps coming on especially after meals. For a certain time the cough is quite dry, and may be said to be ineffectual or hacking, or they may say—for what reason I do not know except for some apparent connection with phthisis—that they have a “tistical” cough, which we find means a dry, hacking cough. After a time there is expectoration, at first mucous, and then purulent. Often indeed expectoration is only seen on first waking, having collected in the alveoli during the night and causing cough in the morning in consequence. Perhaps during the rest of the day the patient has little trouble with it. At the same time he notices, and will probably draw attention to the fact that he is losing flesh, and that is a symptom which always causes alarm. At the same time night sweats are complained of. After a time the patients notice that their breath is gradually getting shorter, that the appetite is poor, especially for breakfast, and that they get various forms of dyspepsia, a sort of discomfort after

meals, and flatulence. Another symptom which is sometimes very pronounced, especially in young females, is vomiting. Sometimes that is the first symptom which draws attention to the illness, and is not by any means unusual in the early stages of phthisis. Whether it is due to some implication of the vagus it is a little difficult to say. At this time the question is probably put, "Are you feverish at night?" They will probably deny this, but if their temperature is taken a slight rise will generally be found. It is well to give directions as to what time the temperature should be taken. It should be taken at eight in the morning, and the usual time at which it is taken in the evening is eight or nine. If only those two hours are selected, one is very apt to miss the most important rise of temperature. It not infrequently happens that in early tuberculosis the temperature is highest at four or five o'clock in the afternoon, and if I suspect that any of my patients are suffering from pulmonary tuberculosis I always suggest that the temperature should be taken between these hours. All the symptoms I have mentioned come on very insidiously, and taken one by one are of practically no importance, and may be due to anything. But when we get this series of symptoms, more especially the rise of temperature and the loss of weight and increasing cough, we should, of course, suspect pulmonary tuberculosis. If a patient comes with these symptoms we inquire into his previous history. We may have a history of repeated attacks of bronchial catarrh. The patient may say he is always catching cold on the slightest provocation, but when the chest has been carefully examined, especially in the young, we may find that the trouble is not in the chest at all, but in the naso-pharynx; very likely the patient has adenoids. Or we may hear that the patient has had pleurisy, and has never been well since. There is no doubt that in a great number of cases of so-called idiopathic pleurisy the cause is tubercle; and in several cases, after tapping the chest of a case of apparently idiopathic pleurisy and examining the deposit of the fluid, tubercle bacilli have been found. This is an important point as regards life assurance, whether a previous attack of pleurisy may have been of a tuberculous nature.

Another form of commencement is what used to be known as "phthisis ab hæmoptœ." But it is

far more probable that the hæmoptysis is a blessing in disguise, and is the symptom which draws attention to the disease, which otherwise would not have been recognised until a later date. Cough is always present sooner or later. Another symptom to which our attention may be directed is pain in the chest. There is nothing more fallacious than pain in the chest, more especially if it be on the left side, and especially in female patients. But if a patient does complain of persistent pain in the chest, the chest should be carefully examined to ascertain the cause of the pain.

Now we come to what are known as the physical signs to be detected in a case of pulmonary tuberculosis, and what conclusions may be drawn from them.

First of all as regards inspection. Very little local trouble can be diagnosed by inspection in the early stages; we may find the chest flattened, or we may find a chest altogether deformed, but it does not at all necessarily follow that the patient has pulmonary tuberculosis. After the disease has gone on for some time we get local contractions; but that does not come until a later period, and does not concern us in considering incipient disease. Another point, which is often omitted but is very important indeed for detecting the disease early, is palpation in order to ascertain the tactile vocal fremitus. But now we must proceed with caution, for we must remember here that normally the tactile vocal fremitus is more marked on the right side than on the left, and in considering these cases we put aside for the moment the question of basic disease, the majority of cases commencing at the apex. In examining the apices in all cases of suspected disease one should never omit this palpation. If the tactile vocal fremitus is equal on the two sides that should arouse our suspicions, and further examination should follow. If, on the other hand, the vocal fremitus is distinctly less on the right side than on the left, that of course is of still stronger import.

Next as regards percussion. Sometimes the first alteration is noticed over the clavicles; you get a change of note by tapping the clavicles more quickly than by percussing any other part. In percussing I think there is nothing so good as the two fingers. By practice one gets what is known as a feeling of deficient resistance. This deficient

resistance is extremely difficult to learn. At first sight it would seem that by deficient resistance is meant that to the pleximeter finger one side seems more firm than the other. But the real meaning is that the difference is felt in the percussing finger; that is to say, we find that in order to get out the same sound we have to use more effort on the one side than on the other. Again, you have to notice the difference between the two sides, because sometimes the percussion note is normally higher pitched on the right side than on the left. So from a very slight alteration like that we cannot, from the percussion note alone, form any definite conclusions. Sometimes the change of note is first noticed in the supra-spinous fossa. Then as regards auscultation, here again you must understand what is normal. It is not at all infrequent to find that the respiratory murmur on the right side is more highly pitched than on the left side, approaching almost to the bronchial. I have frequently seen cases in which students have diagnosed pulmonary tuberculosis because they have found "bronchial breathing," as they call it, under the right clavicle, and yet the history of the patient and the subsequent course of the case showed that there was nothing whatever the matter with the chest. Therefore because we find bronchial breathing under the right clavicle, that sign alone is not sufficient to enable us to make a diagnosis of the disease. The first alteration from the normal is that the respiration becomes irregular and rather harsh; we get harsh, wavy breath-sounds. Expiration becomes more marked than in the normal, and in judging of the respiration much more stress must be laid upon expiration than on inspiration. The first change, then, is wavy breath-sounds under one apex; if it is under both apices we can draw no conclusion from it. In nervous people we are likely to get this sign under both clavicles, but when it occurs under one clavicle only it is a suspicious sign. The next alteration of the breath-sounds is that they become more feeble and more faintly heard on one side than on the other. Especially is that suspicious if you hear feeble breath-sounds on the right side. It is not until we get to this stage, that is to say, feeble breath-sounds, that we get any adventitious sounds. When the adventitious sounds first appear they are as fine crepitations, chiefly at the end of inspiration. These crepita-

tions are not so fine as those which we hear in croupous pneumonia, but more like a dry crackle. They are different to those crepitations which occur later on in the case when disintegration of the lung tissue is going on. Sometimes at this stage they are called clicks, sometimes fine crepitations, and they are especially heard after coughing. Sometimes the crepitations do not occur until the patient has given two or three pretty hard coughs.

Now as regards diagnosis. The point to remember in diagnosis from physical signs is that we must not trust physical signs alone, but, in words of the late Sir Andrew Clark, we should have "the assemblage of signs and progression of symptoms." I have hinted at one of these signs already, namely, bronchial breathing under the right clavicle. That of itself is not sufficient to make a diagnosis; we must have several of such signs together. We must have an alteration in the tactile vocal fremitus, we must have alterations in the breath-sounds, and we must have adventitious signs. And even if we have all three, there comes the difficulty in the diagnosis that we may be dealing with a simple alveolar catarrh, which may pass off entirely. On the other hand, the catarrh may be tubercular. Then, in addition to an assemblage of signs, we must have a progression of symptoms, without which we cannot absolutely diagnose pulmonary tuberculosis. It is in these doubtful cases, where we see the patient only once, that the examination of the sputum becomes so extremely important. Here I would add that a positive examination of the sputum is everything. If a competent observer finds tubercle bacilli in the sputum we may be sure we have to do with pulmonary tuberculosis in some part of the respiratory tract. Very sad mistakes have been made on the part of the examiner, due to not keeping the glass perfectly clean. I have seen a diagnosis of pulmonary tuberculosis made where investigation has proved that it was tuberculosis on the part of the examiner only—that is to say, that his cover-glasses were not clean, or in some way the sputum had got contaminated. I do not think the number of bacilli found makes much difference; it is simply a matter of their presence or absence. Negative results are of very little value indeed. I remember in one case that I examined the sputa nineteen times, on none of which occasions did I find any tubercle bacilli.

Still we were certain that pulmonary tuberculosis was there, and on the twentieth examination I found the bacilli. At the twenty-first examination I could not find any, and after that the patient died. Post-mortem we did find pulmonary tuberculosis; it was of the miliary type. Of course if we have a succession of negative examinations, each inspection with a similar result renders the diagnosis of consumption less probable.

I have mentioned simple alveolar catarrh, and that simple alveolar catarrh may sometimes have tubercle engrafted upon it. Any signs at the apex, such as rhonchi, should lead one to entertain doubts, and the case should be watched. Still, all the physical signs mentioned may occur in simple alveolar catarrh, and it is simply by the progress of the case and the examination of the sputum that we ascertain whether the patient is suffering from tubercle.

There is another class of case which is often brought to the physicians, and which sometimes gives rise to difficulty. It generally occurs in a girl of eighteen or nineteen years of age who has noticed that she has been getting thinner, and more particularly that she is getting paler; she loses strength and has a bad appetite, and suffers from indigestion. But what frightens the friends is that on the pillow in the morning blood is found, or on first waking she expectorates a little. It is not uncommon for anæmic girls to have a slight exudation of blood from the gums which tinges the saliva. This exudation is so slight in the day that it is not noticed, but at night it collects and makes the early morning saliva blood-stained, or dribbles out of the mouth and is seen on the pillow. It is very difficult in these cases to assure the friends that there is nothing worse than anæmia.

Another form of case which gives rise to error is that in which hæmoptysis comes from the throat. I have seen several cases in which I have felt certain that the hæmoptysis has not come from the lungs, but from the throat, and on examining the throat I found a large number of varicose veins. But it has only once been my lot to actually see the blood oozing from one of these veins. One day I was examining a throat of this sort; I had made the patient hawk by the use of the laryngoscope, and there had been a great deal of strain put on the throat. I then asked a colleague to examine the throat, and we saw a drop of blood oozing out

of one of the veins; we put in a piece of cotton wool and convinced the patient that the hæmorrhage did come from the throat.

On the other hand, the occurrence of hæmoptysis may be of the greatest value to the patient, for, as I have already said, it is to him a blessing in disguise. All the other symptoms may be exceedingly slight, and then he may suddenly expectorate blood. That is, therefore, a sign which very often draws attention to the disease before it has reached a very advanced stage. Of course hæmoptysis alone is not sufficient to establish a diagnosis.

A few words as to prognosis. I have referred to the age and the state (whether married or single). Social position is an important point for this reason; if a patient has fair means, and can therefore adopt the most suitable measures for treatment, his chances of recovery are very much better than are those of the individual who has to continue his sedentary occupation.

The type of pyrexia is important, but I have not time to go into it now.

As to the treatment of incipient pulmonary tuberculosis, of course for success in practice we, as doctors, have to look at the disease not only from our point of view, but from the patient's point of view also. Probably it will inspire a great deal of awe and wonder to hear doctors discussing which form of pulmonary tuberculosis it may be, but what the patient wants to know is, whether he is going to get well, and what is the best way of treating him? Well, as to treatment, various drugs have been proposed, each one for the time being vaunted as a specific for the disease. A great number of years ago a certain mixture was introduced into the pharmacopœia of this hospital (carbonate of soda, gentian, and hydrocyanic acid). Since then many drugs have been tried, but we have been obliged to come back to this old prescription. In the present state of our knowledge I am afraid we must say that it is practically impossible to hope to destroy the tubercle bacillus, when it has entered the lungs, by means of drugs; we have to try to make its habitat unsuitable for it to live upon; we have to try to benefit the patient's condition as far as possible, to increase his appetite, and assure that the products of digestion are properly assimilated—that is to say, we must treat a tuberculous patient through

his stomach. The mixture above referred to certainly produces an increased appetite, and is extremely useful. There is no doubt certain tar products, such as creasote and guaiacol, are also of very great use in treating pulmonary tuberculosis. I very much doubt, however, whether the beneficial effects are due to any germicidal action on the tubercle bacilli in the lungs; I believe their action is to destroy in the stomach and intestines those microbes which cause flatulence and fermentation of food. I think carbonate of soda, nux vomica and gentian before meals, and guaiacol and creasote after meals in small quantities is a good treatment for early tuberculosis. Some advocate big doses, but few people can tolerate them.

I must draw attention to what has become the fashionable, and not only fashionable but correct treatment of phthisis—namely, the open-air treatment. In the 'Practitioner' for this month is an article, in his characteristic style, by Sir Samuel Wilks on this subject. After giving his reminiscences as to various forms of treatment which have from time to time been adopted, he mentions Diogenes, and quotes that worthy's saying, "Get out of my sunlight," and suggests that might very well be applied to pulmonary tuberculosis. That is really what our treatment consists of, to a very great extent—namely, letting the patient be in the open air, in baths of sunshine, as much as he possibly can. Fresh air (Sir Samuel maintains) is *the* treatment of pulmonary tuberculosis; without that no drugs are of any use, and therefore, following the example of our German neighbours, the patients should be made to sit out in the air winter and summer. They must be protected from cold and draughts, and there is no doubt patients do far better in that way than by any other treatment, especially if they have various stomachics to aid digestion.

Another novel treatment is as regards the treatment of the pyrexia in early cases. Our general rule used to be when the patient's temperature went up to say 102° or 103° to put them to bed and keep them on slop diet with quinine, anti-pyrim, and so on, to bring down the temperature. Now it is proposed to put them to bed, and then literally stuff them with food as long as the temperature remains up, giving them twice as much as any ordinary individual in spite of the temperature.

It is maintained that the results of this treatment go to show that the temperature of pulmonary tuberculosis is in this way lowered more quickly than by any other means.

It is satisfactory to find that in England several establishments have been formed for this open-air treatment of consumption. There is one in Bournemouth, and several doctors are adopting it. There are others in Norfolk. Now should this be done in sanatoria, or can any doctor carry it out for himself? For the ordinary individual it is better, I believe, to be done individually, so as not to bring a number of patients together. Of course in sanatoria the doctor understands the patients thoroughly and sees them frequently, which is a benefit. Very careful supervision of the diet, &c., is necessary for the first two or three weeks, after which the patient can be left more alone. Of course, owing to the "sample" form of climate usually experienced in this country, we have to take very great precautions to protect our patients from rain and cold winds; consequently a sort of house has been arranged to hold two or three patients which rests on swivels, and the open side can be turned to protect from the rain or winds, or turned to the sun.

[Cases were then demonstrated and examined.]

Polyneuritis as a Sequel of the Pasteur Prophylactic Injections.—Professor Darkschewitsch, of Kasan ('Neurologisches Centralblatt,' 1898, No. 3; 'Deutsche Medizinal-Zeitung,' July 11th), adds two to the reported cases of paralysis following the Pasteur method of preventing rabies. In the first case it began in five days after the treatment had been completed, and was accompanied by pains and impaired sensibility. Both upper and lower limbs were affected, and there was muscular atrophy of the hands. There was reduced reaction to both the faradic and the galvanic current, with tenderness of the muscles and of the nerve-trunks. In the second case was right-sided facial palsy in a week after the last injection had been given, and two days later the left side of the face also was affected. The electrical reaction was the same as in the first case. In both instances there was polyneuritis. Their course was favourable.

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ON IDIOGLOSSIA.

With Special Reference to a Case in which it was associated with Pseudo-hypertrophic Paralysis.

BY

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IN TWO PARTS.—PART II.

Nature and causation of idioglossia.—Idioglossia seems to be simply an exaggeration of defective modes of speech, which are extremely common in minor degrees,—without being regarded as in any way morbid.

For instance, the sound of "s" is commonly rendered "th" by ordinary lispers, Jews or Gentiles; occasionally as "t," more rarely as "k." Thus I remember a certain hairdresser who used to mystify all new customers by remarking, "Kamkoo, kir, oo kave, kir?" when they entered his shop, until they realised that he merely desired to know whether he should shampoo or shave them. His s's were all k's, but he had no other marked defect of speech. R is often represented by "y" or "w." Rabbit becomes "yabbit" or "wabbit." Sometimes R has an "l" sound. This is especially noticeable amongst the Chinese, but is not uncommon amongst English.

At times amongst the lower classes the custom of mispronouncing vowels as well as consonants appears to spring up, become universal, and then gradually to die out.

Most of Dickens' characters drawn from low life substituted v for w; but the error is now practically extinct. In our own time we can all remember a period when the vowel sounds were substituted for each other, a became "ai," o "ow," y "oi," &c. But this confusion, again, was more common ten years ago that it is of late. More recently there has been a tendency to substitute the sound v for th, mother, father, Thursday, became "muv-ver," "farver," and "Fursday,"—with adults and children alike. Oddly enough, what is regarded as

a vulgarity of pronunciation in one age becomes fashionable in another; for instance, the termination "ow" becomes "er"—"fellow" is "feller;"—or final g's are dropped—"shilling" is "shillin." Thirty years ago these abuses of the Queen's English were only heard amongst the uneducated.

It is unnecessary to enlarge on the vicissitudes which the letter h has undergone.

All these common defects of speech are but minor instances of those which obtain in idioglossia.

Idioglossia is only a comprehensive example of them all.

Predisposing causes of idioglossia.—Wyllie (p. 135) concludes that "lalling" upon a single consonant is not entitled to be regarded as an indication of defective intellect, although persistent "lalling" upon many consonants is always very strongly suggestive of such a defect.

In the case of imbeciles it is only natural that wide-spread defects of articulation should be present, but it will always be found in such cases that not only articulation, but syntax and grammar are in fault. An imbecile is mostly a crystallised infant, and his powers of language stop short when his intellect ceases to grow. His vocabulary is limited to a few words, and he makes little use of inflections. His sentences are primitive in form. In idioglossia, on the other hand, the vocabulary is extensive, inflections are used, and the grammatical form of sentences is correct. Every sentence is complete, and each syllable of a sentence is represented by a separate sound.

Mental condition in individual cases of idioglossia.—One of the patients of Dr. Hale White and Mr. Golding-Bird, aged 9 years, had acquired a character for sharpness and ability; the other, aged 10½ years, had always seemed an intelligent lad.

Dr. Taylor's patient aged 8½ years had an intelligent face, and understood everything that was said to him. His palate presented rather a high arch, but he was otherwise well formed.

Dr. Hadden did not regard any of his cases as being deficient in intelligence. Indeed, the great improvement which some of them showed after a systematic course of treatment proves that they could not have been so.

Dr. Colman says (op. cit., p. 1420), "Although the children are often intelligent and quick, the

difficulty of making themselves understood gives other people the impression that they are idiots."

My own patient is bright and quick in understanding, but, as already mentioned, he cannot concentrate his attention for more than a brief space of time. He soon tires, and looks bewildered and worried; then ceases to make any effort to speak properly when pressed to do so. This may be partly due to lassitude occasioned by his physical condition (pseudo-hypertrophic paralysis). But it also depends on the mental confusion induced in this and other cases by prolonged effort. My second case exhibited it in a marked degree if one attempted to regain her attention after it had wandered.

Dr. Hadden mentions that one of his patients would begin to write well at dictation, but after a time would go on writing up and down strokes with no meaning, or, at all events, words which would suggest the language which he usually spoke. He also got confused if he attempted to speak quickly. This was apparently so in Dr. Taylor's case.

I conclude, therefore, that although not actually unintelligent, the subjects of idioglossia have some inherent inability to make prolonged use of the faculties of attention and observation which are necessary for the acquisition of correct speech.

Family antecedents.—Several of the cases occur in members of the same family. In some instances there is a family history of insanity.

The father of one of Dr. Hadden's patients died in Colney Hatch. Two uncles of another were insane; in his third case the father's brother did not speak until seven years of age, and the patient's cousin on his mother's side stammered slightly.

The father of Dr. Hale White's and Mr. Golding-Bird's three patients was said to have spoken badly until the age of twenty, when he was cured by some American process. His mother was in a lunatic asylum. A paternal uncle of the children talked indistinctly many years ago, but subsequently he spoke distinctly. Their mother was a nervous, emotional woman.

A cousin on the mother's side of Dr. Frederic Taylor's patient was an idiot.

In my own case, however, there is no family history of insanity, imbecility, or any other nervous

disorder, except pseudo-hypertrophic paralysis in the present generation.

Yet I think it must be more than a coincidence that a family history of insanity obtained in so many cases. Its significance has yet to be seen in association with idioglossia.

Other diseases and conditions associated with idioglossia.—Many of the patients are healthy and sound, but Dr. Colman met with infantile hemiplegia in one case, and heart disease (? congenital) in another.

Some of the patients are left-handed, and others not. One is said to have been ambidextrous. This was the case with the maternal grandmother of my patient.

Part played by defective audition in producing idioglossia.—Dr. Hadden mentioned in his paper (op. cit.) that "the evolution of speech depends on integrity of auditory perceptive centres." At the discussion on Dr. Hale White's and Dr. Taylor's paper ('Med.-Chir. Trans.,' March, 1891) Mr. Spencer Watson and Dr. Langdon Down attributed the condition to defect of hearing short of deafness. Mr. Spencer Watson suggested that adenoids and naso-pharyngeal complaints might account for defective utterance. Such complaints may certainly lead to indistinctness of speech, but will not alone account for idioglossia. My own patient is quite free from any disease of the sort. In none of the cases was hearing anything but acute so far as mere detection of sounds is concerned.

Absence of musical ear.—I have already laid stress on the singular absence of musical ear in my own case. With the exception of Dr. Colman, who mentions that one of his patients "sang fairly well," none of the writers on the subject allude to the point. Yet I think it has an important bearing on the nature of idioglossia.

If the patient has not the ear to discriminate between spoken sounds, it is probable that he will not appreciate differences between musical sounds.

It will probably be found that people who have literally no ear for music in the sense that they do not appreciate distinctions between sounds, have learnt to speak on the oral and visual method rather than on the auditory. This implies greater exercise of concentration and observation than is needful in those who learn to speak by ear. The

acquisition of their own as well as foreign tongues is usually slow, although by sheer hard work they may learn to converse with considerable fluency, yet with a barbarous accent, in languages used abroad.

Good musicians, on the other hand, are almost invariably good linguists. There may be apparent exceptions to the rule, but they admit of explanation.

Many people profess to have no ear for music, yet have no difficulty in articulation. The late Dean Stanley, although a fluent speaker, was accustomed to say that he did not know one tune from another.*

Charles Lamb, if his "Chapter on Ears" † is to be taken seriously, and not as merely good humoured badinage at the expense of his musical friends the Novellos, might be another instance in point. Yet on his own showing he was singularly susceptible to the emotional effects of music, which he actually disliked.

Many are like Charles Lamb in this respect; not a few are absolutely indifferent to the charm of music, and so have never taken the slightest pains to cultivate it, but it by no means follows that an ear for music is completely lacking in them as in my patient.

Indeed, they may possess a far finer and more correct ear (if they had chosen to train it) than others who profess to be fond of music. The latter often only like the rhythm of a tune, but the richest harmonies which accompany it are disregarded, or, it may be, unheard by them. They enjoy a cornet or violin solo, but would willingly dispense with the band. To the musician these

* M. C. Simpson ('Many Memoirs of many People,' p. 28) says that her father Nassau, senior, was absolutely without ear, and had to be told when "God save the Queen" was played.

† "I have no ear. . . . I even think that *sentimentally* I am disposed to harmony. But *organically* I am incapable of a tune." "Scientifically I could never be made to understand (yet have I taken some pains) what a note in music is, or how one note should differ from another. Much less in voices can I distinguish a soprano from a tenor." "I am constitutionally susceptible of noises. A carpenter's hammer in a warm summer noon will fret me into more than midsummer madness. But those unconnected unset sounds are nothing to the measured malice of music. I have sat through an Italian opera till for sheer pain and inexplicable anguish I have rushed out into the noisiest places of the crowded streets, to solace myself with sounds which I was not obliged to follow, and get rid of the distracting torment of endless, fruitless, barren attention."—'Essays of Elia,' 1884 (Macmillan), p. 52, *et seq.*

are the swine before whom he casts his pearls. However, they readily pick up simple melodies and distinguish one from another, and so cannot be absolutely devoid of ear.

Another apparent exception is in the case of semi-imbeciles, who have often a keen appreciation and a quick ear for music, but whose powers of speech are limited to a few ill-pronounced words. We have already mentioned that the speech of imbeciles is that of infants, and bears only a superficial resemblance to idioglossia. Such cases are not in the same category. Lack of intellect prevents the imbecile, and lack of ear prevents the idioglossic from speaking correctly.

I see no reason, therefore, for doubting that idioglossia in my patient's case depends on defective audition, in the sense that distinctions between sounds are not appreciated or not heard; further, that the absence of musical ear in his case is in accordance with such a view. I have already mentioned the evidence that his visual memory is faulty, and the possibility that his auditory memory is so too, whilst the difficulty in concentrating his attention for more than a short time, which he evinces, and the mental confusion induced by prolonged efforts at concentration, are largely accountable for the perpetuation of his peculiar form of speech.

A similar explanation probably holds good for all cases of idioglossia.

The other conclusions which have been drawn as to the nature of idioglossia are—

(1) The term idioglossia is legitimate as representing the condition of a well-marked group of cases.

(2) Idioglossia is neither a spontaneously invented language nor an example of atavism, nor a "sport" in language.

It is not to be confounded with baby language nor with the language of imbeciles, although it bears a superficial resemblance to them. It is not evidence of weakened intellect, but may possibly indicate hereditary taint of insanity.

(3) It is an exaggerated form of minor and extremely common defects in speech.

It does not depend upon malformation or imperfection of the motor organs of speech, but probably is chiefly due to congenital deficiency of audition—not amounting to deafness.

As such it has no more pathological basis than

has the absence of a liking for sport, or a taste for art, or a sense of humour.

Treatment.—The chief point to be borne in mind is that in all cases hitherto recorded—I believe without exception—correct articulation is possible when the child's attention can be gained sufficiently to make him imitate the mechanism necessary for production of various sounds. If his eye wanders, his ear is no help to him. He must therefore be taught, as the deaf-mutes are taught, to imitate by eye what he cannot do by ear. Also in order to learn the mode of production of sounds such as gutturals, the mechanism of which is not obvious to the eye, the sense of touch must be used. One of the child's hands should be placed on the teacher's throat, and the other on his own, whilst the desired sounds are uttered.

It is first necessary to ascertain which sounds are most faultily produced. Then monosyllabic sentences should be constructed, in which the sounds occur both as initials and terminals—as Dr. Colman suggests,—and the patient should be made to reproduce them by the aid of eye and touch.

It is best that the sentences should be meaningless, otherwise the child will learn them with ease in his own jargon, and will cease to take pains to repeat them correctly.

Few medical men have leisure to devote the time and patience necessary for the treatment of these cases, so instruction must be left to a relation or friend intelligent and persevering enough to carry it out on the lines laid down. In some cases it may be desirable to isolate the patient from all but his instructor. Relapses are not uncommon when teaching is lax and intermittent.

In addition, exercises should be devised to train the visual memory, *e. g.* the patient should be taught to draw from memory simple, and by degrees more elaborate figures, after carefully studying their copy.

Probably the ear may be cultivated by systematic musical training. Many people become creditable executants—vocal and instrumental—by industry, although by nature they have no musical proclivities. After much trouble my patient learnt to render a recognisable tune; but when seen two months later, instruction having lapsed meanwhile, the faculty had apparently deserted him.

As his life will necessarily be a short one, it is not desirable to distress him by further efforts to improve his speech. But the success which follows systematic instruction in other cases, and the serious bar to prospects in life occasioned by idioglossia, warrant prolonged endeavours to overcome this *cacœthes loquendi*.

Chronic Intestinal Indigestion.—In the course of an extended article upon this subject, S. Henry Dessau states that for children suffering from this complaint all starchy foods, except in the most limited quantity, should be forbidden, unless one of the amylolytic ferments, as diastase or takadiastase, is previously added. Coffee and tea are to be condemned, also fats and sugars, except in the smallest amount. Meat will be found to agree better for a while until the digestive functions are restored, but it should be eaten in a solid form, not as soup or extracts. Hydriatics in the form of warm wet packs, followed by a cold sponge douche of 60° F. to the spinal column and abdomen, with brisk friction, are admirably indicated to promote oxidation of tissue and elimination of waste matter. Cold wet compresses to the abdomen, followed by massage along the line of the colon, will be found a valuable aid in constipation of infants. Rectal irrigation with Kemp's tube and water at 110° F. is a most efficient means of relieving the attacks of abdominal pain. In infants the principal indication for the use of drugs will be to relieve constipation and flatulence. For this purpose calomel and bismuth are the only drugs worth considering. As an intestinal antiseptic the bismuth may be given in the form of the subnitrate, subgallate, or beta-naphthol bismuth. After dentition the author prefers the use of a tonic laxative, such as cinchona and nux vomica, with senna or cascara sagrada.

Amer. Journ. of Obstet., July, 1898.

FRÄNKEL has lately read a paper on bronchial asthma. He had had the opportunity of a careful study upon a patient, a man 60 years of age, who had been suffering for the previous year from attacks of bronchial asthma, and who died in one such attack. Therapeutically Fränkel would recommend a subcutaneous injection of hyoscyamus hydrobromate.

Medical Record, August 6th, 1898.

CHAPTERS FROM THE TEACHING OF DR. G. V. POORE.

No. III.

GENTLEMEN,—I think it is advisable, before getting to the more technical parts of the course, to give a sketch of the legal relations of our profession.

It seems tolerably clear that in the early stages of society the functions of the physician have always been conjoined with those of the priest. Hippocrates, the father of medicine, was probably a priest in the temple of Æsculapius at Cos. In the Hebrew literature we find that the priestly class conjoined with their sacerdotal functions those of the physician and the medical officer of health. And so when you come down to later times in this country we find the same thing, the priest and the physician joined in one person. The earliest record that we have in this country of the physician is the doctor of physic mentioned by Chaucer. There is one line in Chaucer's account of the doctor which is very often quoted, and it is sometimes quoted (wrongly) to prove that as a profession we are apt to be a little sceptical on matters theological. Chaucer says of his doctor: "His studie was but litel on the Bible." Now the meaning of that is this, that his medical duties had diverted him, as it were, from his strictly clerical duties. The early priests who devoted themselves to medicine made a lot of money, and those who devoted themselves to surgery made more; so that Pope Innocent III, who lived at the end of the twelfth century, found it necessary to issue an edict that priests should not draw blood, because he found that they were going after filthy lucre. This edict of Pope Innocent III caused an early separation between physicians and surgeons. Physicians still continued to be culled from the ranks of the priests, but the surgeons came from the ranks of more practical men. Now mark the difference. The physician, or rather the priest-physician, was naturally brought up to regard authority as absolute; and just as the priest was not allowed to question the truthfulness of his Scriptures—using the word Scriptures in its usual sense—so the priest-physician was not allowed to question the authority of the medical scriptures. The medical scriptures

consisted of the works of Hippocrates and Galen and others, and there is in the early records of the College of Physicians the account of a Fellow who doubted the authority of Galen, and who was made to recant upon his knees. That is an interesting fact, and one which effectually prevented any progress. On the other hand, it must be remembered that the early physicians were largely men of high education, and from the earliest times were graduates of Oxford or Cambridge, and were men of culture in its widest and best sense. It has always been the practice of the College of Physicians to look largely to the culture and character of its members and fellows, and I take it that we owe an immense debt to the College of Physicians, because it has raised the level and kept up the level of medicine in this country. I have no hesitation in saying that the medical ethics which pertain in this country will compare favourably with those which pertain in any other country.

As society developed the necessity for physicians and surgeons was more and more felt, and we find that early in the sixteenth century, in the reign of Henry VIII, a Medical Act was passed (1511). The Medical Act was passed because it was found that ignorant people and wise women were practising medicine, not always to the benefit of their patients. And so it was enacted that nobody should dare to practise medicine who had not been examined and approved by the bishop of the diocese. There again you get the link with the clerical profession; but it is right to state that the bishop of the diocese was allowed to call to his aid persons having knowledge of medical and surgical matters. In 1518 Henry VIII granted a charter to the College of Physicians, and the founder of the College of Physicians was Linacre, a man of great classical learning. He had studied abroad, and was one of the leading men of the time, certainly one of the leading scholars of the time. But Linacre was a priest, and not only was he a priest, but he was a pluralist, and his clerical revenue was very large indeed. But let us be thankful for it, because he made good use of it. He established the College of Physicians, and founded valuable lectureships both at Oxford and Cambridge. Those who were associated with Linacre in the early days of the College of Physicians were also priests. Of course with the Reformation this state of things came gradually to

an end. Still I may remind you of the fact (as evidence of the relationship of our profession to the Church) that the Archbishop of Canterbury still retains the power of granting an M.D. degree—M.D.Lambeth—and we need not grudge him that power, because he can only give the degree now to persons who are legally qualified. He occasionally gives the degree to a medical missionary who has deserved well of both the clerical and the medical professions. As far as I know there has been no abuse of this power, and I should be sorry to see that little link with the past destroyed.

So much for the physicians; how about the surgeons? How did they come? Surgery must have been wanted in the world earlier than medicine. Epidemics of disease were largely looked upon as visitations of the higher powers, visitations against which it was useless to struggle. But wounds were regarded probably differently, and the man who could staunch a wound and preserve a limb must very soon have been appreciated. War must be regarded as the mother of surgery. And certain it is that some of the earliest and best known surgeons were men who accompanied the armies of their sovereigns abroad. In civil practice surgery took its rise from those who I suppose were accustomed to use sharp weapons, and occasionally to inflict sharp wounds; and it need not surprise us that the surgeons are an offshoot of the barbers. One of the oldest surgical institutions in this city is the Barbers' Company, formerly the Company of Barber-Surgeons, that dates back to the reign of Edward IV. Formerly, as you know, a barber was accustomed to bleed; and sometimes even now, in country places, you see the barber's pole (painted in alternate stripes of red and white, to represent the bloody limb and the white bandage), and the bleeding dish shaking in the wind. That old sign of the barbers must remind you that the surgeons are sprung from them.

The Charter of the College of Physicians dates from 1518, and twenty-two years later Henry VIII gave a charter to the Barber-Surgeons; and in the Barber-Surgeons' Hall in Monkwell Street, which is not far from the General Post Office, there is a very famous picture by Holbein of Henry VIII giving the charter to the Barber-Surgeons. Soon after this the Company of Barber-Surgeons recog-

nised a distinction between those who practised surgery and those who practised barbery. In 1745 the surgeons separated from the barbers, and established a hall of their own in the Old Bailey.

"Honour to whom honour is due." I have said something in praise of the early physicians; now I will say something in praise of the early surgeons. We owe the early surgeons a deep debt of gratitude. The early physicians, practising mainly by the light of their "scriptures," did not trouble themselves very much about the arcana of the human body; but the surgeon who had to operate soon found that a knowledge of anatomy was absolutely necessary. In the charter given by Henry VIII in 1540, a clause was put in that the surgeons might have the bodies of a certain number of criminals for anatomical purposes. The surgeons in their hall in Monkwell Street, in the City of London, established an anatomical theatre, where they gave anatomical demonstrations. They engaged for that purpose a physician of the highest attainments, who had been abroad to learn anatomy at the great school of Padua. Among the early anatomists in this country was Dr. Caius, the founder of Caius College. Another man who read anatomy to the surgeons was Dr. Scarborough, a very eminent man in his day, and also a great mathematician and friend of Harvey. You will find in Pepys' 'Diary' a graphic account of how Mr. Pepys went to see a demonstration in anatomy at the Barbers' Hall. The early surgeons having set the example as regards anatomy, the physicians very soon followed, and Queen Elizabeth gave an addition to their charter that they might have the bodies of criminals. Then Caius also got permission to have anatomical demonstrations at Caius College, Cambridge.

So much for the surgeons. Let us now say a word about the apothecaries. The apothecaries originated in a different way; and it is not surprising to find that the Apothecaries' Company is an offshoot of the Grocers' Company. The grocers were people largely concerned in importing what we should call colonial goods. With these they imported a good many things which were used in medicine as well as in cookery, and it is not surprising that as medicine advanced the number of drugs multiplied prodigiously, and that a separate company was organised for their supply. The Apothecaries were established in the early

days of James I, in the hall close to Blackfriars Bridge, which no doubt many of you know.

In its early days the College of Physicians exercised very great powers. They had absolute control over the practice of medicine in London and a certain radius round it, and they often put irregular practitioners in the lock-up. The surgeons might not administer drugs, and there is a case on record of the President of the College of Physicians putting a surgeon in prison for administering a purgative. Those early physicians undertook a great many prosecutions of quacks—and it is interesting to note that these prosecutions often came to nought, because then, as now, the quacks had influential friends among the governing classes.

The early physicians set their faces also against unprofessional conduct; and if a man's conduct and morals were not what they ought to be, they drummed him out of their society, and held him up to public obloquy.

Next we find apothecaries, who were originally druggists pure and simple, prescribing. For a long time a battle raged between the physicians and the apothecaries, but ultimately the apothecary became a practitioner who supplied medicine, and a very useful person he was and is.

The profession remained in a rather chaotic condition until the middle of the present century. It was in 1858 that the first Medical Act was passed. That Medical Act was amended or supplanted by the Medical Act of 1886, and we are now governed by the Medical Act of 1886. By that Act it is ordained that no person can be registered as a legally qualified medical practitioner unless he has passed a qualifying examination in all three branches of the profession—medicine, surgery, and midwifery. The qualifying examinations may be conducted by any university or corporation capable of giving a diploma, or any combination of two or more universities or corporations, provided the conjoining bodies are in the same part of the United Kingdom. You know that the Colleges of Physicians and Surgeons in London have combined and formed a conjoint board for conducting qualifying examinations. The efficiency of the examinations is supervised by the General Medical Council; that body appoints inspectors to attend the examinations, and if in the opinion of the General Medical Council the examinations are not stringent enough, the

General Medical Council has the power to report the fact to the Privy Council, and the Privy Council has the power to ordain that the diploma given by such defaulting body shall be suspended until it amends its ways.

If a corporation has not the power of examining in the three branches of the profession, it is unable to give a qualifying diploma. The Apothecaries' Company had the right to examine in medicine and midwifery, but not in surgery, so that it could not grant qualifying diplomas, its attempts to effect a combination with other corporations having failed. The Privy Council in accordance with the Act of 1886 intervened, and gave them permission to appoint examiners in surgery; thus the Apothecaries' Company can now give a qualifying diploma. The Medical Register is controlled by the General Medical Council, and the General Medical Council is composed as follows:—there are three Crown nominees for England, gentlemen nominated by the government. It is a very important thing that a certain number should be nominated by the government, because then they are not delegates in any shape or form, and they can take a perfectly independent view of every question. Then there are delegates from the Royal College of Physicians, the Royal College of Surgeons, the Society of Apothecaries, the Universities of Oxford, Cambridge, London, Durham, and Manchester (Victoria University). In addition there are three members elected by the registered practitioners of the country. The total representation for England is thus fourteen. For Scotland there is a Crown nominee, one elected by the practitioners, and one each from the Royal College of Physicians of Edinburgh, Royal College of Surgeons of Edinburgh, the Faculty of Physicians and Surgeons of Glasgow, and the University of Glasgow, the University of St. Andrews, the University of Aberdeen, and the University of Edinburgh. For Ireland there is one Crown nominee, one elected by the practitioners, and one delegate from the Royal College of Physicians of Ireland, the Royal College of Surgeons in Ireland, the Apothecaries' Hall of Ireland, the University of Dublin, and the Royal University of Ireland. The total strength of the General Medical Council is therefore thirty. When it meets, the Council elects one of its members as President. The late President, as you know, was Sir Richard Quain,

and the present President is Sir William Turner, the distinguished Professor of Anatomy in the University of Edinburgh.

The main duty of the Council is to guard the entrances to the profession, and it also has the duty of expunging from the Register persons who have been guilty of certain offences. To these duties is added that of compiling the Pharmacopœia. The corporations have bye-laws of their own, and many of them expunge from their lists those who are guilty of unprofessional conduct. If a man has been convicted of any gross crime his name may be expunged by the corporation which gave him his diploma, and then the General Medical Council has the right to consider whether his name shall be expunged from the Register or not. The Council is now very much alive to the importance of suppressing unqualified practitioners in every shape and form, and has lately issued an edict that unqualified assistants shall no longer be employed by qualified men. Now the General Medical Council is a useful body, but we must not expect too much from any governing body; and while we have an eye to proper qualifications and honest practice, we must also keep the other eye fixed upon our liberties both as medical practitioners and citizens.

Medical men have certain privileges, and are exempt from certain duties. They are not bound to serve on juries, and they need not take unless they choose certain corporate and parochial offices. Our profession is a very exacting one, and a man who is liable to be called suddenly to those who are seriously ill would have to set aside his public duties. By an old Act of Parliament medical practitioners were exempt from serving the office of constable, watch, scavenger, and overseer, and from bearing arms and serving on juries; and the same thing has come down to our own time.

Unless a man be legally qualified—that is, unless he holds a qualifying diploma and be upon the Register—he labours under certain disabilities. The first disability he labours under is that he cannot recover debts for professional services. No member or fellow of the College of Physicians is allowed to recover debts in a court of law. It is one of the greatest privileges ever bestowed upon us. It keeps us out of courts of law, and insures that the physician gets his fees at the time he is consulted, which is an enormous advantage.

Therefore the unqualified man is on a level with the highly qualified man in respect to the recovery of debts. I do not think it is a great disability to labour under, because the unqualified man takes care to get his money before he gives his advice. Another point is that a man who is not legally qualified cannot hold certain offices under the Crown, and cannot, without danger to himself, give a certificate of death nor a certificate of lunacy. With regard to death certificates, I remind you that you are bound to give them. I see that there was a case the other day in which a practitioner refused to give a death certificate because his bill had not been paid. He lost his case, because you cannot demand payment for giving a death certificate. If you have knowledge of the cause of death you are bound to give a certificate; the law will order you to do so. With regard to giving a death certificate, let me say that it is a very important duty. It is an important duty as regards the public, and it is an important duty as regards yourself. And let me implore you never to give a death certificate hurriedly and without filling up the counterfoil. The forms of death certificate are supplied to you gratis by the registrars. You should always fill up a death certificate quite truly. Always give it serious thought, because certificates of death are the groundwork of medical statistics, the value of which cannot, I think, be over-estimated.

NEUROTIC ECZEMA.

L. DUNCAN BULKLEY, of New York ('Journal of the American Medical Association'), gives the following interesting description of this disease:—Neurotic eczema is particularly interesting to the general physician, because if recognised and rightly treated, not only are the results commonly very satisfactory, but the patient also receives much improvement in general health by the measures calculated to benefit the eczema. Neurotic eczema should always be recognised as the signal-flag of danger, and should never be passed over with attempts to gain relief from local treatment alone, which can only be of minor and temporary benefit.

The influence of the nervous system in the pro-

duction of skin lesions of various kinds has long been recognised, and is now abundantly established both by pathological and clinical proof.

Neurotic eczema is seen in both sexes and in all ages, although it is much more common in certain periods of life than in others. In infancy it is frequently observed in connection with cutting of the teeth, fresh eruptions occurring as each new tooth presses on the swollen and tender gum. In childhood it is less common, but may appear with each occurrence of nerve-strain, whether from over-work in school or over-excitement in recreation. The same is true in youth, where the changes of puberty come in as a factor, especially in females. But its most frequent time of occurrence is during that active period of existence between twenty and fifty-five years of age, when the strain and burden of life falls heavily on so many, and when the strongest constitutions too often show many evidences of breakdown both in the nervous system and in other directions.

Various forms or phases of nerve disturbance are seen in connection with neurotic eczema, and they may be considered under the following heads: (1) Neurasthenia, or nerve exhaustion; (2) nervous and mental shock; (3) reflex phenomena—(a) of internal origin, (b) peripheral; (4) neuroses—(a) structural, (b) functional.

Neurotic eczema does not differ very greatly from other forms of the eruption, but a trained eye can generally suspect the true character of the case. The eruption is apt to come first upon the hands and face, less commonly on the feet. But from its starting-point it may extend over large surfaces, and after scratching or irritation by treatment may present quite the features of gouty eczema.

Neurotic eczema on the hands is very apt to exhibit vesicles, but on the adult face the eruption is quite as likely to assume and maintain the erythematous form without vesicles, and often without moisture unless scratched. The groups of lesions of neurotic eczema have a tendency to be pretty sharply defined in more or less herpetic patches, which may present mainly solid papules, or when torn a raw surface. The areas affected early in the eruption are not apt to be very large, but the eruption is composed of a number of patches of aggregated lesions.

Neurotic eczema is intensely itchy, and the patient will often scratch where there are no appa-

rent lesions, and so develop the eruption in one place and another. The scratching indulged in is commonly of the most severe kind, it seeming almost impossible to reach the seat of the trouble until the deepest portions of the epidermis are reached. The spasms of itching are sometimes fearful and utterly uncontrollable.

Treatment will often tax the patience and skill of the physician to the utmost, and the broadest principles of medical knowledge and judgment will often need to be put in operation and maintained if the patient is to have permanent benefit. The treatment includes both constitutional and local measures; the former are essential, the latter are helpful. Arsenic undoubtedly finds an important place in the treatment, but should never be relied on alone, and the author seldom administers it in the form of drops, the Fowler's solution so generally given by the profession for every case. Iron, nux vomica or strychnine, quinine, the phosphates, ergot, oils, and many other remedies may come into play in the management of the disease. In occasional cases much benefit will be obtained from digitalis in strengthening and regulating the capillary system, and so improving nerve and cellular nutrition, while other cases will require the sedative action of aconite in free doses.

A few words may be added in regard to the attempt to give relief to the itching and securing sleep in neurotic eczema, by the administration of internal remedies, hypnotics. While the eruption is in an aggravated condition it often seems almost impossible to effect much by this means. Opium and its preparations only aggravate the itching, and if sleep is secured it is of an entirely unrefreshing character. Codeia seems to be the least injurious of them all. Sulphonal and trional in large doses will sometimes be effective, but are often followed by further nervous exhaustion, in the end aggravating the complaint. Phenacetine in full doses, repeated in an hour or so, will sometimes prove most effective, and antifebrine, with hot water and a little whisky, will often secure refreshing sleep; in milder cases urethan in one-gramme doses is effective. Tincture of gelsemium, given in repeated and increasing doses every half hour, has in some instances proved most serviceable in the author's hands, as has cannabis indica. The bromides have relatively little effect, although when combined with a very small dose of mor-

phine and aconite have at times proved very valuable.

The diet must always be carefully directed. The one article which contains the elements necessary to nourish the nervous system and proves of the most signal advantage is milk, if properly taken. This should not be used at all with meals, nor after or in connection with the least particle of food, otherwise it undergoes caseation and may embarrass the liver, which is too often at fault in these cases. The author's plan is to have the milk taken warm, pure and alone, one hour before each meal, and also at bedtime, if sufficient time has elapsed for the stomach to be perfectly empty, which is at least four hours after a hearty meal. When the digestion is sluggish, and the milk meets the late products of digestion, it is curdled, and then its digestion requires some considerable time and we do not get the benefits desired. But if it can be taken on an absolutely empty stomach, when the alkaline tide has already set it, it is absorbed almost immediately and affords a refreshment to the whole system, including the nerve elements, which can hardly be obtained in any other way, and which must be observed to be thoroughly appreciated.

The local treatment should be soothing and protective, and the mildest applications should be made first. Zinc ointment with 1 or 2 per cent. of carbolic acid or creosote, or with 5 to 10 per cent. of ichthyol, or tincture of camphor, is always a safe and generally beneficial dressing, if well and thoroughly applied. But to be of service it should be kept thickly applied, spread on lint in most places and bound on firmly. In the acutely inflamed, and especially in the erythematous forms of the eruption, there is nothing better than the well-known calamine and zinc lotion, freely sopped on many times in the day. Ichthyol in watery solution, 10 to 20 per cent., freely bathed on the part, often gives great relief, or if the skin is too dry it may be used in oil in the same strength.

On more chronic patches the permanganate of potassium, 2 per cent. solution in water, painted over the part will sometimes arrest the itching very well; it may either be used alone, or the calamine and zinc lotion may be sopped on after the surface has dried, or an ointment may be applied if the skin is at all hard.

In the erythematous eczema of the face a tannin

ointment, 1.95 to 3.9 grammes to the 31.2 grammes with 2 per cent. of carbolic acid, is effective; a mixture of camphor 1.95 grammes to 31.2 grammes of zinc ointment also forms a good antipruritic. In still more chronic states the tar and zinc ointment, as recommended by the writer many years ago, when thickly spread on lint and bound on, will often serve admirably to control the itching.

The use of very hot water for a brief application, followed by an appropriate ointment, should never be forgotten. In old cases of eczema of the scrotum the effect of this treatment is sometimes very remarkable. Menthol will often prove a valuable addition to ointments, in the strength of 2 to 4 per cent., with about half as much carbolic acid, which latter serves both to heighten the antipruritic effect and also to overcome some of the chilly sensation caused by the menthol.

Medicine, August, 1898.

Reflex Iridoplegia.—It would seem that unilateral pupillary disorders in general, including reflex iridoplegia, are as a rule the result of interruption in the centrifugal portion of the reflex mechanism on the same side, and it is more than probable that bilateral reflex iridoplegia is due to the same conditions affecting both sides. This statement does not include loss of the light reflex occasioned by disease of the optic nerve or retina, which is usually associated with blindness. The following conclusions seem warranted:—(1) That unilateral reflex iridoplegia is a condition which may arise in tabes or parietic dementia, being confined to one side for an indefinite time before the other pupil becomes similarly affected. (2) That it is also found in cerebral syphilis, and may be permanently limited to one eye. (3) That it often occurs as a remote result of disease of the third nerve or its nucleus, and may be the only demonstrable clinical evidence of a pre-existing third nerve paralysis. (4) That it is always indicative of central-nerve degeneration involving either the oculo-motor nucleus or its efferent branches. (5) That it is generally of syphilitic origin. (6) That the lesion producing unilateral reflex iridoplegia is situated in the centrifugal portion of the reflex mechanism.

Leszynsky, *N. Y. Med. Journ.*, Aug. 6th, 1898.

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ON MITRAL DISEASE.

A Clinical Lecture delivered at the Hospital for Consumption and Diseases of the Chest, Brompton, March 2nd, 1898,

By CHARLES ARKLE, M.D., F.R.C.P.,
Assistant Physician to the Hospital.

LADIES AND GENTLEMEN,—You probably know that the mitral valve is attacked more often than any of the other valves by inflammation—endocarditis as it is called—and consequently diseases of this particular valve are perhaps of more importance for your consideration than almost any other cardiac lesion. If you take any thousand cases of heart disease, you will probably find that some 500 of them are affections of the mitral valve. The aortic valve you will find affected in something like 350, and the remainder will be found to be affections of the right side of the heart. In dealing with mitral disease, and beginning at the commencement of the affection, one has to remember that in its origin it is primarily an inflammation of the endocardium of the heart at that particular site, and of course the commonest cause of all of this inflammation is the rheumatic poison; it occurs as a result of an attack of "acute rheumatism." Scarlet fever is a very common cause of this affection, and chorea is another, the latter being very intimately connected with rheumatism. You will remember in passing that chorea is often followed or accompanied by rheumatism, and rheumatic affections are often associated with chorea, and that in cases of chorea you can find, in the great proportion of cases an intimate connection with the rheumatic poison. I shall have something to say later with regard to rheumatism as it is found in children, but I will say at once that you must bear in mind very strongly that rheumatism in children frequently passes absolutely unnoticed; that children will be brought to you, and you find marked cardiac lesions in them, without any serious suggestion from them or their parents that they have ever

suffered from anything like rheumatism. Without asking leading questions you try and find out whether there has been anything like rheumatism, and it is often almost impossible to get any trace whatever of a history of acute rheumatism. The explanation is that these children have some affection which is practically as dangerous as acute rheumatism in an adult, but they run about with it, and it is recognised by nothing more than a few "growing pains," as they are called, in the limbs. Of the other acute specific diseases, measles and chicken-pox much more rarely cause endocarditis; scarlet fever is far in advance of these diseases, and second only to acute rheumatism and chorea. Now, given an inflammation of the mitral valve, or the structures in its immediate vicinity, what is it that you generally find? Of course post-mortems in such cases are rare; an attack such as this does not often destroy life, but in the few cases of chorea and acute rheumatism which I have seen to terminate fatally, and examined subsequently, I have found uncommonly little. There may be just a few granulations, a little redness and swelling of the edge of the valve, and so on, but nothing very striking to the eye. But you do find that the endocarditis is not a general affection of the whole lining of the heart; it is generally confined to one or another place; it may be the mitral valve, it may be the aortic valve, but it is not a general inflammation. Then the parts which suffer most are obviously those which are liable to the most strain, and that of course explains why the mitral valve suffers more than the aortic, the mitral valve having to bear the whole force of the ventricular systole, whereas the aortic valves have only to bear the force of the arterial recoil. The parts of the valve which you find most affected are the parts which come most into contact, not the free edge, but a little above the free edge in the case of the mitral valve; and in the aortic valves it is not the lunula, but a part a little below that. Remember that in looking at the semilunar valves, especially the aortic valves, the small holes which you find in the free flowing edge are quite compatible with health; they do not mean disease or rupture or anything of that kind, but "fenestration" of the valve, as it is called, is of no significance whatever. If you examine an inflamed valve from a case of rheumatism or chorea under the microscope you find the usual

signs of inflammation; a certain amount of swelling and a certain amount of exudation, leucocytes here and there throughout the tissues,—points upon which it is unnecessary to dwell further. In life, however, what happens is that the blood-current deposits fibrin on these inflamed parts, and you get the formation of vegetations which may play an important part in the after-history of the case. A point which strikes one as of very considerable importance is, what is it that determines, in any particular case, whether you are going to get a case of mitral constriction, stenosis as it is called (obstruction of the mitral orifice), and what is it that determines whether you will get a case of mitral regurgitation? This distinction is of primary importance, because, as you probably know quite well, mitral regurgitation is a comparatively simple form of heart disease, whereas, as I shall have to tell you later, mitral stenosis or constriction is a very serious one. I suppose that whatever it is that determines what form of valvular defect you are going to get all happens in the early stages of the disease. At one stage or other of the affection the tissues are evidently softened; at one stage we see the chordæ tendineæ occasionally ruptured, or attenuated, and we see perhaps an aneurysm appearing in the valve itself. Such considerations show, I think, that at some stage or other the tissues of the valve must be soft. If this extends to the mitral ring as a whole, I suppose it is likely that we shall get a case of regurgitation, for the cusps of the valve will not come together if the mitral ring is softened and dilated.

Such a case will probably be one of mitral regurgitation. In a very considerable number of cases, however, you find that the inflammatory process passes this stage, or perhaps never reaches it; then the tendency of the process is to go on to thickening of all the structures, a thickening and shortening of the chordæ tendineæ, so that in well-marked cases you may hardly find the tendinous cords at all, the papillary muscle seeming to be inserted on to the valves themselves. This sort of cicatricial change leads, as you know, to narrowing of the mitral valve, and is a very much more serious condition, I think, for the patient than dilatation. Therefore one of the questions I think you may profitably consider is, what causes one case to go on to regurgitation and another to stenosis? Further, the mitral valve may be affected in those

more malignant cases of acute ulcerative endocarditis; they present no new features clinically, except that of course they are very much more serious, and they very much more often lead to a fatal termination. Their detection generally lies in the fact that they resist treatment, the temperature is intractable, and they run a pyæmic course. Always have your suspicion about such cases when they resist the ordinary treatment, and their temperatures keep up and look pyæmic. But between the simplest variety and the most malignant there are all gradations. Sometimes you see a case where the valve is covered with very exuberant granulations. Sometimes you examine a case where there are regular ulcers or abscesses in the substance of the heart muscle or in the neighbourhood of the valves. Between the very worst and the most favorable there are all gradations. I need not tell you that in the malignant cases these vegetations, when they are dislodged, are of a very malignant and infective nature, because they are infested with micro-organisms; and if they form emboli in the organs they very commonly lead to abscesses, which run, like the disease generally, a pyæmic course. In one group of cases of mitral disease you find conditions resembling the atheromatous changes which you get in the larger blood-vessels; I mean those cases where the process is an extremely chronic one, and where you get thickenings, fatty softening, and very often the deposition of large quantities of salts, producing in reality a calcification of the valve. These seem to form a distinct group, and the lesions are very often of a constrictive nature. In some cases there is an enormous deposit of calcareous matter in the valves.

Let us take mitral regurgitation first. It is the simplest and least harmful of the two great lesions of this valve. I suppose a mitral regurgitation murmur is generally the first murmur that a student of medicine recognises. The results of mitral regurgitation are probably familiar to you, and I need not therefore say very much about them. Of course the first thing that happens when a reflux takes place through the mitral orifice is that the left auricle dilates. The second thing which happens is that the left ventricle dilates, and subsequently, if matters go well, hypertrophies. The third event is that the right ventricle dilates and hypertrophies. And last of all, if the case is doing

well, the right auricle does the same. One looks for and generally finds this course of events as the patient recovers and as the lesion becomes established. Such an event is generally recognised as compensation, and one hopes that this compensation will be good, and so the case goes on for a variable length of time—it may be for a great many years—the condition not perceptibly shortening the patient's life. But if untoward events happen, this compensation may break down, and then you get another sequence of events, which I shall have to speak of later.

Now when the patient presents himself here for examination he is very often quite unconscious that he is suffering from anything wrong with the mitral valve, and perhaps the first inkling you have of it is when you come to listen to the heart, so little does it affect his comfort or appearance. But in almost every case one has to remember that the patients probably come for some reason, that they are not feeling up to the mark, and one has to bear in mind the kind of symptoms that they come with. They come generally when their compensation is just beginning to break down, and as they present themselves you are perhaps struck with a certain amount of cyanosis about these patients; it may be only slight lividity of the lips or darkness of the finger nails, or it may be much more pronounced. These patients generally have a certain amount of shortness of breath. Another very common sign is clubbing of the finger ends; but remember that you get well-marked clubbing in other diseases, and especially in cases of consumption. It is said, however, that the clubbing in the two diseases differs somewhat; in mitral disease the finger nails are not curved, whereas in phthisical patients the clubbing is associated with a good deal of incurvation of the finger nails, said to be due to the loss of fat which takes place in this wasting disease. In children remember that the first sign of clubbing is a certain "shininess," as they call it, at the base of the finger nails, this being about the earliest sign of clubbing which you can find. Their further troubles probably consist of a certain amount of bronchitis, or it may be congestion of the lungs, and there may be blood-spitting, and finally perhaps a certain amount of palpitation or irregular action of the heart. These are the early troubles which generally bring the patients to the hospital. If the compensation has

failed to a further extent you find all these symptoms and physical signs are more exaggerated. Instead of simple cyanosis you will probably find evidence of extensive venous engorgement. You may find a very irregular action of the heart, or extreme palpitation. Instead of simply slight lividity there may be more intense blueness, associated with a certain degree of sallowness, giving a very characteristic earthy appearance to the face. There may be swelling of the feet, or there may be general dropsy, which has begun in the feet and afterwards extended to the body cavities. If you come to examine the internal organs you will probably find a big liver, an enlarged spleen, a certain amount of catarrh of the stomach, and sickness, and there may be diarrhoea, pointing to a similar condition in the intestines, with diminished flow of urine, which is probably albuminous.

The physical signs of a case of mitral regurgitation I need say very little about; you are probably quite familiar with them all. You will probably find signs of an enlarged heart and of a dilated heart when the case comes before your notice; so that on inspection there will be a certain amount of bulging of the præcordium, with displacement of the apex beat to the left, which palpation will confirm, and show you an hypertrophied and dilated heart, and probably a heart of very irregular action. In the modern works on the subject of heart disease I have been somewhat struck to find that a great many people seem to say that the pulse of mitral regurgitation is a regular one. Authorities seem to differ a great deal, but I feel myself that if there is any characteristic about the pulse of mitral regurgitation which is at all constant and reliable, it is that it is irregular, not only in force but in rhythm. Some writers further say that the pulse of mitral stenosis is irregular, but this is exactly the reverse of what I have observed. Authorities seem to differ on both points. Percussion will reveal the enlarged heart, and auscultation will give you your apical systolic murmur. There are one or two points which ought to be noted about this latter. You can describe the qualities of this murmur as you find it in different cases—very variable, blowing or musical, and so on, and you may add any further distinctions you like. But are there any other murmurs which you can mistake for it? If you hear a murmur at the apex of the heart which is conducted out into the left axilla and heard at the

angle of the left scapula, can you be fairly certain that that means mitral regurgitation? There is one murmur which you must remember, and that is the hæmic murmur. If you hear a murmur at the apex, and you think it may be a hæmic murmur, you have got to look elsewhere and see if there is any confirmation. In these cases you will probably find a hæmic murmur at the base of the heart, or possibly a murmur systolic in time over the whole præcordium, and you will probably hear a very good venous hum. This, coupled with the appearance of the patient, must keep you on the right path as to whether the murmur is a hæmic or an organic one. A point of some importance to observe is to listen whether there is an accentuated pulmonary second sound. Then you sometimes get at or about the apex an exocardial rub. You know that about the middle of the anterior surface of the right ventricle post mortem, and very often about the base of the heart, you may sometimes see what is called a milk-patch. Sometimes it is called a "corn" of the heart. It is where the ventricle strikes against the chest wall, and I think it is conceivable, and indeed highly probable, that in those cases you sometimes get a very short rub, which may resemble a systolic murmur at the apex. It is an interesting patch, and you see it on a great number of hearts. An interesting point about it is that you seldom see it on the hearts of children. If you do see it on the heart of a child it is more often on the left ventricle, and you may be perfectly certain that the child was rickety, the explanation being that the chest in rickets is so commonly deformed that the apex of the left ventricle gets this rub, and the white patch appears in this situation. Now there is a murmur sometimes heard—systolic in time—at the apex and produced it is said by the little piece of lung which overlaps the heart. The ventricular systole expresses from this little bit of lung a certain amount of air, and it gives a whiffing kind of murmur. I think that this is perfectly right, and the best way of checking it is to ask the patient to stop breathing. It is heard best on inspiration, when the lung is fairly fully inflated with air; then the heart's impulse gives you this little jet, which is really produced in the lung, but has the same rhythm as the heart.

The points to remember in coming to a diagnosis are first of all this systolic apical murmur, which you must do your best to separate from any other

possible bruit; the markedly accentuated second sound, which is a very important point; and finally enlargement of the heart transversely.

You will see here specimens of hearts the subject of mitral regurgitation and of mitral stenosis, and of both conditions together; you will see also examples of ruptured chordæ tendineæ, and of vegetative endocarditis, which latter I have said have all the gradations between high malignancy and comparatively simple inflammation, but in the matter of obstruction or regurgitation, or both conditions together, it will be quite easy for you to come to an opinion.

We now come to mitral stenosis. Narrowing of the mitral orifice is a very different disease, I think, to mitral regurgitation. It is a disease which occurs in early life, and is generally found in females. It is said to be the heart disease of little girls. The explanation of this seems to be that females are more subject to rheumatism than males; that in addition to that there are the effects of chorea, with which girls are more often affected than boys. Out of eighty cases of mitral stenosis sixty-three were in females. Then as to chorea, half the severe cases of chorea examined had evidence of organic heart disease, and one third of these lesions were those of mitral stenosis. These figures seem to agree with the explanation that it is the greater liability of girls and women to rheumatism and chorea which makes the disease more common in the female sex.

Now let me emphasise again the importance of remembering how easily rheumatism is overlooked in children, for it has a very important bearing on treatment. I am perfectly certain that children have rheumatism while they are running about, and that their rheumatism is as dangerous to the heart as if they had all their joints full of fluid, and were confined to bed. Perhaps a little girl is brought to you, and you listen to the heart and find she has got well-marked mitral stenosis; it is extremely interesting, without putting any leading questions, to inquire from the mother whether there has been anything like rheumatism, and you will generally find you will get a certain history of "growing pains," and sometimes a more marked history than that, such as pains in the joints and so on, but there will be no history of confinement to bed, or of any severe or definite illness.

There are two common varieties of mitral stenosis. One you know is where the process has been somewhat chronic, and you get a so-called "button-hole" mitral valve. That is where the segments are all fused together and contract up, so that you just get a sort of diaphragm between the auricle and the ventricle, with a narrow button-hole slit leading from one to the other. A second kind is where you get a "funnel-shaped" mitral, the chordæ tendineæ being greatly thickened and sometimes fused together, the papillary muscles perhaps being inserted right on the edge of the valve, so that you get a funnel-shaped aperture passing from the auricle to the ventricle. This aperture may be extremely small so as to admit only a thick probe.

These are the common varieties of change which are found in mitral stenosis. You have next to decide whether the case is one of pure mitral stenosis, or is there regurgitation along with it? This is important, because it makes all the difference in the world to the condition in which you will find the heart. In most of the hearts which have been brought out for our inspection, if not in all of them, I should say they were the subjects of mitral regurgitation as well as mitral stenosis, but in the little girl E. D—, and in the young woman M. B—, the physical signs show almost certainly only a narrowing of the orifice, without evidence of regurgitation. Think what the heart is in a case of pure uncomplicated mitral stenosis. Begin at the mitral valve, which is narrowed. The left auricle is most certainly dilated and hypertrophied; instead of being a flimsy, thin-walled structure it is thick and fleshy. What is the ventricle? It is a small one. The left ventricle in a pure uncomplicated mitral stenosis looks like a mere appendage compared to the right ventricle. There has probably been very little blood in it, and it has had no increased work to do, the aortic valves being healthy; it has probably had less work because there is a less quantity of blood to throw into the unobstructed aorta. But you find a different state of affairs with regard to the right ventricle; that is greatly hypertrophied and dilated; it has had increased work to do to get the blood through the lungs, and so you find a large right ventricle, and possibly in later stages an incompetent tricuspid valve and a large right auricle. In some of the text-books it is said that the left

ventricle in mitral stenosis is hypertrophied, but I am quite positive that that is not so; and you have only to use your eyes to see that in any uncomplicated case of mitral stenosis the ventricle is not a big one, and there is no reason why it should be.

A point which ought to be mentioned in connection with mitral stenosis is that it is a very common cause of embolism. Clots form in the heart and become detached, get into the circulation, and form emboli in the different organs. That is one of the points which makes mitral stenosis a much more serious disease than mitral regurgitation. The clots seem to form most commonly in the dilated auricular appendix; as far as can be made out this is the favourite seat for them. Some stagnation of blood occurs in the tip of the auricular appendix, to commence with, while occasionally you may sometimes find a large ante-mortem clot moving freely about in the distended auricle. And remember that these ante-mortem clots may form beneath the columnæ carneæ and elsewhere, and that the first part of the clot which you get lies there, and very soon becomes surrounded by a layer of fibrin; then the layer of fibrin which surrounds the original clot becomes decolourised, and fresh layers are added, and you get a laminated clot. To my mind "lamination" in a clot is the best evidence that the clot was ante-mortem. I do not lay much stress on adherency of clot. When the clot is laminated that shows better than anything else that the fibrin has been deposited during life. But the central part of the clot does not become decolourised, though it very often softens, and in such a clot you may get a broken-down substance containing leucocytes (detectable under the microscope) and some purulent-looking fluid—not true pus—surrounded by layers of fibrin. One of these ante-mortem clots breaking up will give you emboli in all distant organs in the greatest possible profusion. If the clot gets in a cerebral vessel the results are often disastrous, while they account for the spitting of blood and the blockage of arteries in different parts of the body which one so often finds connected with this disease.

The physical signs you are probably fairly familiar with. Remember that in mitral stenosis very often you get a good deal of deformity of the chest—some precordial bulging, and one cannot help noticing that many of the cases are associated

with pigeon breasts. By palpation you get a thrill which, if it is well marked and can be easily timed, is the key to the whole disease. Mitral stenosis is one of the few heart diseases which you can diagnose by palpation. A thrill felt at the apex in a somewhat limited area, presystolic in time and finished by the impulse, can only mean mitral stenosis. We can speak quite definitely about it. Then on percussion it will be found that the cardiac dulness is increased to the right, and that bears out my remark about the small left ventricle. The presystolic murmur which you detect on auscultation (for you will remember that a thrill is only a murmur which can be felt) is of very great interest indeed. While a student of medicine recognises his mitral regurgitant murmur with fair readiness and with considerable precision, it is often very different in the case of the presystolic murmur. It is often very hard to persuade students that the murmur they are listening to is presystolic. They hear a murmur which is definitely followed by the second sound, so they say, and in answer to your question they will say it is systolic, it is a case of mitral regurgitation. Then you ask them to time the sound, by placing the finger on the heart's apex-beat, telling them it is the first sound, not the second. The only true heart sound they hear is found to be synchronous with the impulse, and they then see their mistake. The first sound of the heart becomes remarkably altered in mitral stenosis, so that without due care you may easily mistake it for the second. The first sound gets almost exactly the clear valvular ring of the second sound, and the explanation is, I suppose, that the ventricle is contracting on only a small quantity of blood. It is an imperfectly filled ventricle, and probably goes off rather suddenly. I think that is the best explanation of it. Whatever the correct explanation is, it is often of very great difficulty to distinguish it from the second sound. It is easy, however, if you put your finger at the side of the stethoscope, when you will notice that it is synchronous with the impulse, and that brings the murmur into its proper position in the cardiac cycle at once. In a certain stage of mitral stenosis you do not hear the second sound at all at the apex. The reason which has been advanced for this is that the ventricle only throws a small jet of blood into the aorta, and the semilunar valves

do not fly back with their usual force. Therefore I ask you to remember that in some cases of mitral stenosis at one period of the disease you get a very greatly altered first sound, an absence of the second sound at the apex, at times a diastolic murmur which does not run up to the impulse, then a definite presystolic murmur, and last of all absence of murmur altogether. These latter are perhaps the most difficult cases to be sure of. If they come to you without a history, you get a very curious galloping rhythm; no murmur or normal sound to tell you where you are, no impulse perhaps to guide you, but this short altered first sound to mislead you, and these cases are very difficult to identify. It may be that when you put such cases on digitalis that the murmur will return, but I think they are the most difficult cases of all. I think you get such a state of things more often in mitral stenosis than in any other cardiac valvular disease, and that fact must be your best guide. The danger in mitral stenosis which one must remember is that the process shows a great tendency to recur, and the lesion is a progressive one. Aortic regurgitation often terminates life quickly; but patients may go on for a number of years while suffering from this affection, whereas mitral stenosis seems, from its progressive nature, to be often of even more doubtful prognosis. When you look at a diseased heart, you will find that the parts most affected are those subject to the greatest strain, and this constant strain on a narrowed valve seems to keep up a slight inflammation, and this in turn leads on to contraction, until at last a point is reached—at least in mitral stenosis—when compensation, heart muscle, and everything breaks down. In addition to that you know that embolisms are more liable to occur in this affection than in almost any other, and this fact increases the risks of a bad termination to a very large extent.

With regard to the treatment of these cases of mitral disease I have not much to say. The point has often been raised as to whether, when patients come to you and are not showing any signs of what is called cardiopathy, you ought to treat them at all. You may rely upon this, that when they come to consult you they come for some reason or another, and even if they are ignorant of what is the precise lesion there is probably something wrong with them. I should say treat them always. Listen to

the lungs and ascertain if there are any rhonchi or any signs of congestion; examine the liver, and see if there is any passive congestion there. But even if the troubles be slight I think it is right to treat their symptoms. As you know, a common drug is digitalis. All I have to say about that drug is that very often you find the infusion of digitalis acts very much better than the tincture. The tincture seems to me occasionally to vary in strength and efficacy, and not long ago all the mitral cases on the tincture in the hospital were doing badly, and on inquiry the sample was found to be rather old. Improvement was very striking on placing them all on the infusion. I think the tincture is liable to alter a good deal by being kept, and I much prefer the infusion. Digitalis regulates the heart, it diminishes the frequency of its beats, it prolongs its diastole, it strengthens its contractions, and it increases the arterial blood-pressure, so that it is really our sheet-anchor in the treatment of mitral disease. Failing digitalis—and it does fail sometimes—I go to strophanthus, which sometimes acts when digitalis fails. Convallaria is another favourite of mine. I am not an advocate of the Schott treatment of heart disease. I think you can do as much as you ought to venture to do without those baths and exercises. I think I have seen instances where the Schott treatment has done a great deal of harm. I much prefer in an ordinary case of mitral disease to follow the advice of Oertel and order gentle exercises on slightly rising ground, but not to go in for gymnastic movements either there or on the level.

Relapsing Perityphlitis.—Dr. H. Kümmel, of Hamburg ('*Berliner klinische Wochenschrift*,' 1898, No. 15; '*Wiener klinische Wochenschrift*,' July 14th), has practised excision of the vermiform appendix, in the stage of freedom from acute symptoms, in a hundred and four cases, without a death. It has not been uncommon for him to find the appendix in a condition that an inexperienced observer would consider normal; but in such cases, instead of being soft and flaccid, it is stiff and firm. The greatest number of relapses take place during the first year—in his experience, a hundred and seven in a hundred and forty-five cases—but the first relapse occurred in the tenth year in one case and still later in four others.

LECTURES ON DEFORMITIES CONSEQUENT UPON INJURIES OF THE BONES AND THEIR TREATMENT.

Delivered at the City Orthopædic Hospital,
March 15th, 1898,

By JOHN POLAND, F.R.C.S.

LECTURE II.

GENTLEMEN,—You will remember that on the last occasion I dealt with deformities of the bones as a result of injuries to the long bones, both in infancy and during intra-uterine life. I also dealt with deformities as a result of juxta-epiphysal sprains, passing on to those which result from that very interesting group of cases, the separation of the epiphyses. I explained to you how frequently these cases are brought before us at this hospital, resembling as they do all kinds of deformity about the joint, and very often mistaken for dislocations. I took first of all the deformities after these injuries as they affected the upper extremity, the clavicle, the collar-bone, the upper end of the humerus, and then the lower end of the humerus. To-day I propose to go on from that point and to explain to you first some of the more interesting features of the injuries as they affect the upper extremity.

Taking first the deformity after separation of the lower end of the radius, this deformity resembles very much in its characteristics Colles's fracture at the lower end of the radius; in fact, I may say that separation of the lower end of the radius in youth almost entirely replaces Colles's fracture in the adult. I shall not go so far as to say that there is no such thing as a Colles's fracture in a child, because there is a specimen in St. Bartholomew's Museum of a fracture at the lower end of the radius with impaction, but I believe that to be the only specimen in existence. What we more usually find is such a condition as we have in this cast (here shown) taken from a case of marked deformity with separation of the epiphyses of the lower ends of the radius and ulna, which came under my notice here on October 12th, 1894. I shall just detail to you the history of this case, as

I believe it to be most interesting, and typical of what we so frequently find in all these cases. I ascertained that the accident had taken place five or six weeks previously. The boy's age was ten years. On September 6th he fell at school five feet while jumping, and put out his left hand to save himself. A point worth recollecting is that a greater degree of violence is required to produce this injury in a child than in an adult. The boy immediately experienced great pain in the wrist, which was much displaced, the deformity being of the same character as when he came under my observation, but at the latter time it was more marked. He was taken to one of the large hospitals in the north of London, where the bones were replaced in position. Posterior and anterior splints were applied and retained for a month. At the end of that time the splints were removed, and the bones were again found to be displaced, quite as much as when he had applied for treatment. When I saw him there was the typical "silver fork" deformity, as the French call it, indicating displacement backwards of the lower epiphyses of the radius and ulna with the wrist and hand, but the epiphysis of the ulna did not appear to be so much displaced as the radius, neither was the hand deflected to one side or the other, as you see in Colles's fracture. That is another distinguishing point between these two injuries to be borne in mind. The styloid processes of the bones were in normal relation with the wrist, but the fragments were very firmly consolidated at this time, and some thickening of the shafts of the bones could be felt on the dorsal aspect upwards from the diaphyso-epiphysal lines. The radial artery was pushed prominently forward by the displaced lower end of the radius in front; the flexor tendons were also very prominent, and around them a largish ganglionic swelling had developed. That again I wish to draw your attention to, because you do not see it in old Colles's fracture in the adult. I have no doubt it is produced by the forward pressure of the diaphysis of the end of the bone. In a child, when the injury first takes place, the diaphysis projects forwards, as I shall show you presently, and you get a very sharp angle in front. That is one of the diagnostic signs of the recent injury. As a result of that prominence forward pressure is made upon the flexor tendons and upon the sheath, and gives rise

to synovitis, and a ganglionic swelling results. The fingers were kept slightly flexed, as seen in the cast. Complete active extension was not possible, but with this exception all movements of the hand and wrist were good. Both the points I have just related are entirely different to what you will see in an old Colles's fracture in an adult. There would be no ganglionic swelling in Colles's fracture, but there would be a general rotundity and stiffness of the wrist. Again, you know how often in Colles's fracture you get stiffness of the wrist for months. In this case a moulded leather wristlet was worn five months and massage was systematically kept up, and at the end of this time the ganglionic swelling was reduced, the flexor tendons were less prominent, and the diaphysial end of the radius was rounded off and less distinct. The movements of the wrist were now perfect. When I saw him three months afterwards there was but little deformity left, and no swelling about the flexor tendons. In October, 1896, there was no distinguishable deviation from the natural outline.

[Drawings were here shown taken from photographs at that time.]

In the moulding process the prominence becomes rounded off, and new deposit takes place from the periosteum along the posterior aspect of the lower end of the radius, and the diaphysis gets rarefied.

The next case is an extreme one, but it shows very well how all the parts get moulded down. I shall detail this case to you because it is so typical of a large number of others. The specimen is described by an Italian surgeon named De Paoli, and is preserved in the Riberi Pathological Museum. It shows separation of the radial epiphysis consolidated in the abnormal position that I have described. The diaphysis is much enlarged, especially on the posterior and inner part, where it presents some rough surfaces terminated by a clean edge, which, commencing below at the outer margin, ascends obliquely upwards on the diaphysis, and terminates on the inner margin at the line of insertion of the interosseous ligaments. Its lower extremity has left the upper surface of the epiphysis almost completely on the inner side. I have taken this picture [shown] from a photograph which this surgeon has produced. The epiphysis, the upper edge of which is very dis-

tinct, and does not show any considerable alteration from the normal shape, has been displaced backwards, and its articular surface looks in such a way that the direction of its outer lateral edge, instead of forming the lateral edge of the diaphysis, makes an angle of 45° with its prolongation below. The antero-posterior section of the specimen gives a clear idea of what has taken place. It will be seen that the epiphysis is only in contact with the posterior part of the diaphysis for a distance of 2 mm. About 45 mm. from the lower end of the diaphysis a compact layer covers the diaphysis for a short distance, gradually thinning upwards, and passes downwards to join the posterior margin of the epiphysis. The normal compact tissue of the posterior aspect of the diaphysis may be traced as far as its lower end. The irregular triangular interval behind is filled up with new osseous tissue, which constitutes the principal bond of union between the epiphysis and diaphysis.

The preparation was taken from a woman aged 20, who had the lower radial epiphysis separated some years prior to her death. The displacement had not been corrected, and the epiphysis had consolidated in this position. It looks, at first sight, as though there had been impaction in the lower end. If you examine closely you will see that the compact layer of the shaft is becoming rarefied, and will disappear altogether in time, so that at this stage the radius presents very much the appearance of a very old Colles's fracture. Here there is no impaction, the triangular interval is filled up by new bone. It shows that the whole pathology is very different from that connected with Colles's fracture. I may add that permanent impairment of motion of the wrist and hand, after separation of the lower epiphysis of the radius, is never seen in young adults. There is none of that almost permanent stiffness of the wrist, fingers, and hands, lasting for months or even years, which you see in Colles's fracture in the adult, although there may be some temporary embarrassment in the very severe cases. The effusion into the synovial shafts is rapidly absorbed, and but little fear need be apprehended of adhesions forming if passive movements are commenced early and systematically performed. In the case I have mentioned the movements of the wrist were slightly restricted, and the ganglionic swelling extended above the annu-

lar ligaments. But at the end of a few months it disappeared and the movements became more extensive; there was no shortening, and only slight prominence at the epiphysal line. Ankylosis of the wrist after these injuries is not produced by extension of the inflammation to the joint, but may ensue in those rare instances where the separation is associated with splitting or fracture of the epiphysis into the joint or in compound separation. Here again you see the pathology is very different from Colles's fracture. Wasting of the forearm and hand is never severe, and if movements and massage had been properly carried out it would have been of only a slight and transient nature. Although non-union of the fragments in this accident has been alluded to by some writers as uncommon, this occurrence is open to doubt; in fact, no authentic case has ever been recorded. Union may be retarded by movements, but non-union never appears to be produced.

As to arrest of growth after injuries and subsequent deformity in the forearm, as in the case of other bones which lie parallel, shortening of one bone from arrest of growth is usually accompanied by marked deformity.

The hand, often a little atrophied, is thrust into the valgus position, the transverse diameter of the wrist is increased. The inclination being to the radial side, the hand makes with that of the forearm a greater or less angle. The ulna appears to be displaced downwards and outwards, the end of the bone being very prominent, but in the great majority of cases the articulation is unaltered. There are several specimens in the museums of London hospitals showing great dwarfing of the radius and deformity of the forearm after what was probably an epiphysal separation. This drawing of a specimen in the museum of the Royal College of Surgeons shows an arrested development of the radius, forearm, and hand after injury in childhood. There are other specimens in the museums of the London Hospital, St. Mary's, and there is one figured by Professor Nicoladoni. These cases of arrest of growth of the radius are not common in practice, but a few cases have been recorded by Goyrand, Poncet, Dupuytren, Clay, Haslam, Holmes, Wright, Hutchinson, Appleyard, Stehr, and Professor Annandale.

Passing now to arrest of growth in injury to the lower epiphysis of the opposite bone—the ulna,—

we find a very different appearance. The hand is in the varus position. The ulna depends almost entirely for its nutrition upon the integrity of the lower epiphysis for growth in length, while its upper epiphysis is of comparatively little importance in this respect. From the examples of arrest of growth it would appear probable that injury to the lower epiphysis of the ulna is not so rare as has been supposed. Arrest of growth from injury to its lower end, as in the case of the other parallel bones—radius, tibia, fibula—therefore gives rise to much lateral displacement or adduction of the hand from the continued growth of the radius. The hand is, as I have stated, thereby thrust into the varus position.

The drawing I pass round is a striking example of this position of the hand, but in this case it is complicated by deformity of the elbow-joint which followed arrest of growth in the ulna in a boy aged eleven years. Other instances are referred to by Ed. Owen, Poncet, Hutchinson, jun., and Goodspeed.

[A drawing was then shown of very much the same deformity after osteitis and suppuration of the lower end of the ulna.]

I now come to deformities after traumatic separation of the epiphyses of the metacarpal bones. The following interesting case of displacement forward of the epiphysis of the metacarpal bone of the index finger was sent to me by Dr. Atkins, of Sutton. The patient was aged fourteen and a half years, the son of a well-known medical man. He injured the right hand in April, 1893, by falling with a stick twisted between his fingers. Separation of the epiphysal head of the metacarpal bone of the first finger, with displacement forward, was diagnosed at the time. Under chloroform the epiphysis was readily replaced backwards into its normal position, but on account of some deformity recurring I was asked to see the case. There was still some tendency for the epiphysis to ride forward towards the palm, and also the same outward displacement. Beyond this there was subluxation of the first phalanx. Chloroform was again administered and the finger placed in good position. In spite, however, of the repeated and careful application of plaster of Paris and other splints, the epiphysis again became displaced forward, but all lateral displacement was entirely corrected. In this position it finally united to the diaphysis, as

you see in the drawing. The result is now that the head of the metacarpal bone is still prominent in the palm. The power of flexion at the metacarpo-phalangeal joint is restricted, and the finger is shorter than its fellow, but its usefulness is scarcely impaired. Mr. Sidney Roland took a skiagraph of the hand three years after the accident, which entirely confirmed the diagnosis of separation of this epiphysis. The space which is seen between the diaphysial end and the first phalanx is probably occupied by fibroid tissue forming some portion of the new articular surface between the bones. The epiphysial discs of the other metacarpal bones are clearly seen in the skiagraph. You will see from these drawings a very similar appearance in a case of another kind which came under my notice last year. It is a dislocation of the phalanx from the head of the metacarpal bone of the index finger, with displacement forward. I show you also a lateral view of the same case. Neither of these conditions—dislocation of the metacarpo-phalangeal joint, or separation of the head of the metacarpal bone—are common. Indeed, these are the only two cases I have met with in my experience.

I shall now pass on to the lower extremity. I want to bring before you specially those cases which have come under my own observation, because I think that will be more useful than if I were to state a number of platitudes. I will take first the deformities of the hip and lower extremity which follow separation of the head of the femur. The usual appearance presented is somewhat similar to that of dislocation forward of the head of the bone, which, as you know, is an exceedingly rare form of injury in a child; it is rare in an adult, but still more so in a child. I have never seen an instance in a child. A more common form of dislocation in a child is backwards on to the dorsum; but even that is rare.

I will now show you a photograph and describe a case of epiphysial separation in a boy aged twelve years. He came under my observation at this hospital on July 20th, 1890, two years after the accident, and was admitted for deformity of the left hip, with a history that on August 30th, 1892, he had been knocked down and run over by a hansom cab. He was taken to a hospital, where traumatic dislocation of the hip was diagnosed and easily reduced, as I was later on informed by

letter. After seventeen days he left the hospital with plaster-of-Paris dressing and crutches. The plaster casing was removed after a month, and at the end of three months he was only able to walk with a limp. This got worse, and in April, 1894, he again slightly injured the hip at play, and at the end of May, six weeks later, he was taken to the same hospital on account of the stiffness. Under an anæsthetic some adhesions were found and broken down. As the stiffness recurred the patient was brought to me at the City Orthopædic Hospital. He was a muscular boy for his age. The left foot was everted, so that the inner side of the foot looked forwards and inwards, and the patella outwards. The muscles of the thigh were wasted, and there was rather more than a quarter of an inch shortening. The great trochanter was very prominent with great thickening in front and behind, especially in front towards the neck of the bone, so that this process felt almost twice as thick as the opposite one. It was half an inch nearer the iliac crest than on the right side; the gluteal fold was lost on this side. The leg was neither adducted nor abducted, and the patient was unable to raise it from the bed; passive flexion of the thigh was only permitted to an angle of 140° with the trunk, and rotation was almost lost. There was a slight amount of rotation inwards, but that produced some pain. Many of these symptoms were very much like those met with in dislocation, even at that time, but other signs existed which are never seen in dislocation. It is important to recognise these because at first it was treated as a dislocation, and afterwards it was brought here with that particular deformity which you see in the drawings [shown]. I showed the case to one of my colleagues here, and he was inclined to think it was an unusual case of tuberculous disease of the hip. Although there were some of the signs of tuberculous disease, with displacement of the femur, there was the history of the injury and the deformity, with stiffness gradually coming on from that time. I manipulated the joint and broke down the adhesions which had formed round the osteophytic processes in front of the joint, and it seemed better for the time. I then let the patient go out of the hospital, but he got steadily worse, all the signs becoming more and more marked, so that four months later I decided upon operation.

By an anterior incision along the tensor vaginæ femoris I excised the greater portion of the head and the end of the neck of the femur, which was rotated forwards, so that its epiphysial surface presented in front. A small portion of the head of the bone appeared to have been broken off and displaced, the rest of the head of the bone having undergone rotation in the acetabulum. The articular cartilage was undergoing fibroid degeneration, as you will see in the specimen. No trace of the ligamentum teres could be found, and it had evidently been ruptured. The neck of the bone was firmly united to the epiphysis. The parts were removed in several pieces. The great trochanter was uninjured, and therefore left intact. After the operation the eversion was entirely corrected, and the limb could be freely flexed, and the patient made a good recovery. I did not remove the whole head or neck of the bone, but left enough to form a good socket to that joint. He had almost a perfect joint afterwards.

In contrast to that case I should like to detail to you another one, a lad whom I showed here at my last lecture. He was twenty-one years of age, and when he came under my care there was great eversion and adduction of the lower extremity, and this I diagnosed as being due to fracture of the neck of the thigh bone within the capsule. You will know that this is a very rare injury in a youth. This patient came to me for deformity twelve months after the injury. He had fallen off a ladder to the ground, a distance of eight or nine feet, on his right side. At the time the pain was rather severe, and he felt shaken, but after a few minutes he got up and walked home, although limping. He had no medical treatment. Four months later he noticed that the hip was growing out, and he began to limp. Shortening appeared to increase until I saw him. There was no pain felt except after a long walk. Now what did you see in this case as the patient stood in front of us? The photographs taken at the time of admission show the attitude very well. The pelvis is much tilted, the right leg is very everted, the great trochanter prominent and situated further back than on the left side. The apparent shortening is very considerable, namely, about three and a half inches. As a matter of fact, there was very little if any shortening. The hip was almost fixed. I came to the conclusion that he had fallen on to his hip and

broken the neck of the thigh bone, and that it had not been treated, but the patient walked about on it, causing the head of the bone to become displaced, and it was evident that there was callus uniting two fragments together. This became more prominent, so that it formed a large mass in front of the hip. The only thing to be done was to remove the head of the bone, because the joint was perfectly fixed. I did so, cutting down by an anterior incision, as in the last case. I removed the whole head and only a small portion of the neck of the thigh bone, which I now show you. During the operation there was found a curious condition, for on cutting down on the head of the bone there existed a large swelling beneath the iliacus muscle, which was really a distended capsule. The articulation was full of blood-stained fluid. In the specimen you can still see the line of the epiphysis. Another curious point is that the ligamentum teres is gone, and the head of the bone is becoming rarefied. The cancellous tissue of this head is disappearing, and the articular surface is becoming fibrous. It has lost its proper nutrition, all that it had was from the periphery of the epiphysial line. The fracture having occurred at this point, there is only that circulation to carry it on. I suppose the head would ultimately have undergone perfect atrophy and absorption. The reason I performed the operation was that the prominent osteophytic mass in front absolutely prevented any flexion of the joint. [The patient ultimately had a freely movable hip-joint without deformity.] Non-union after separation of the head of the femur I believe to be very uncommon. The grave prognosis Van Sweten gives, and the inevitable consequences which are said to follow this injury, may be traced to Boerhaave. Reichel relates the case of a young man, twenty years of age, who had had from infancy a separation of the femoral head. There was no union; a false joint developed, which allowed the man to turn the limb in all directions and to carry his foot easily up to his head. The limb was twelve inches shorter than the other.

KÖNIG relates the case of a week-old baby with genu recurvatum, bent tibia, marked club-foot, flexion of left hip, and constriction of left leg below the knee. Evidently intra-uterine pressure caused the lesions.—*Amer. Journ. of Obstetrics.*

ON TWO CASES OF PERIGASTRIC ABSCESS, ARISING FROM GASTRIC ULCERATION, AND RUP- TURING INTO THE LEFT LUNG.

A Clinical Lecture delivered in the Medical Department
of University College, Sheffield, May 24th, 1898,

BY

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THERE are several different varieties of gastric ulcer; to-day I shall speak only of the simple or chronic gastric ulcer, which occurs chiefly in young women, and is by no means uncommon. The symptoms, apart from those due to complications, vary greatly in severity in different cases. There may be little more than a feeling of discomfort after taking food, so slight that medical advice is not sought; whilst on the other hand there may be very severe pain and vomiting, causing considerable distress. The chief symptoms of gastric ulcer are—

1. Epigastric pain, worse at a definite time after meals.
2. Vomiting, which tends to relieve the pain.
3. Hæmatemesis.

Of these three the most important from a diagnostic point of view is hæmatemesis; at the same time it is well to remember that this symptom may be absent from beginning to end, or that the blood being slight in amount and altered by the acid gastric secretion, it may escape the patient's notice. The more serious complications of gastric ulcer are—

1. Profuse hæmatemesis. Extremely alarming but usually recovered from.
2. Perforation. The gravity of its results varies according to whether it occurs—

(a) Before adhesions have been formed between the outer surface of the stomach and neighbouring parts sufficient to shut off the general peritoneal cavity. Acute general peritonitis—usually fatal.

(b) After sufficient adhesions have formed to shut off the general peritoneum. Then one of three things usually happens, either—

(i) A neighbouring organ becomes the base of the ulcer, and remains so if sufficiently thick (*e.g.* pancreas); or

(ii) A neighbouring organ being thin walled, becomes at first the base and then is itself perforated (*e.g.* colon), thus forming a fistulous opening; or

(iii) The perforation not being over a closely applied organ, or being at the edge of it, there is no proper base to the ulcer, and the contents of the stomach passing through set up a localised abscess which tracks in various directions.

The two cases which form the subject of my lecture to-day are examples of this last condition.

CASE 1. K. C. A.—, single woman aged 24 years, was sent into the Royal Hospital under my care by Mr. Longbottom on December 15th, 1897. She had been quite well until April, 1897, when she began to suffer from pain in the stomach, which came on about one and a half hours after taking food; this was usually followed by vomiting, which relieved the pain. On two occasions she vomited bright red blood, about a cupful each time, the last time about six weeks ago. The history was thus typical of gastric ulcer, and such had been the diagnosis made by Mr. Longbottom. She had had a mitral systolic murmur for some time.

On admission her temperature was found to be 103° , and she complained of some dull aching pain in the epigastrium and left hypochondrium. The next day or two her temperature became normal, but on the 18th December it rose again, and from that time onwards there was a fairly regular evening rise to 102° or 103° , and morning fall to about 100° . On examining her I found a considerable amount of resistance over the epigastrium, and the cardiac impulse in the fourth interspace a little outside the nipple line. On December 21st, at 1.30 a.m., she had a severe rigor with a temperature of 104° , urgent dyspnoea, and great collapse. The temperature rose to 106° at 2.30 a.m., and she then began to expectorate considerable quantities of stinking pus. Later in the morning I saw her, and her condition was then as follows:—"She lies on her left side, where she complains of much pain. The respirations are frequent and shallow. Pulse 130, small and weak. She looks very ill, and is spitting up small quantities of very offensive gruel-like purulent

matter at frequent intervals. Physical examination: the lower part of the left lung hardly moves at all, there is hyper-resonance over the upper part of the left chest in front. Dulness in the axillary region and skodaic resonance at the left base behind. A tympanitic note reaches up in front as far as the fourth space, where the impulse is felt. There is loud bronchial breathing over the left axillary region, cavernous breathing over the base back and front, with well-marked metallic dropping sounds such as one hears over the abdomen."

The diagnosis was made of a subphrenic abscess in connection with an old gastric ulcer and subsequent rupture into the left lung. An exploratory needle was inserted into the left base, but produced nothing. The patient continued much the same, expectorating small quantities of similar altered pus, and having a daily rise of temperature until December 25th, when, as the expectoration had diminished considerably without any improvement in the general condition, I asked my colleague, Mr. R. J. Pye-Smith, to perform an exploratory operation and drain the abscess. This was done on December 25th, and proved more prolonged and difficult than had been anticipated, only a very small quantity of pus being found at the base of the left lung. The patient, who had been very ill for some days, stood the operation badly, and died the following day.

At the autopsy made the following day the stomach was found distended and lying almost vertically from above downwards. It was adherent to the front edge of the left lobe of the liver, over which was part of an abscess cavity. On opening the stomach an oval ulcer one and a half inches by half an inch was found about the middle of the upper or lesser curvature, and running on to the posterior surface. The upper part had perforated completely through, and communicated with the abscess cavity over the front of the left lobe of the liver, whilst the lower half had also perforated, and its floor was formed by the pancreas. The abscess cavity passed at first behind the left lobe of the liver, then turned forwards a little way to the right, but chiefly up to the left, surrounding the cardiac end of the stomach and passing as far as the spleen. Its upper boundary was the diaphragm, which it had perforated, forming a communication with the left lung, which was adherent above to the

diaphragm. It was almost devoid of purulent contents, although none had drained away since the operation; its walls were composed of the various organs and structures mentioned above, and shut off completely from the general peritoneal cavity by adhesions. The heart showed old vegetations on the mitral valve, and was somewhat dilated. In this case the abscess cavity communicated directly with the stomach through a comparatively narrow fistulous track behind the liver.

CASE 2.—A. S—, a single woman aged 18 years, was admitted into the Royal Hospital under my care on March 30th, 1898. The menses began when she was fifteen, but since that time she had seen nothing. When thirteen she had inflammation of the bladder and pain on micturition. For the last six months she has had pain on micturition. For three years she has suffered from indigestion, with pain and vomiting after food, for which she has been treated for the last few months. Both her parents died of consumption. About the second week in March she fell downstairs and hurt her left side badly, so that she had to go to bed for five days. After this she got up and tried to work, but was unable to do much. On March 23rd she began with constipation, which lasted till the 29th. On March 25th she had acute pain in the left side, and took to bed. On March 28th she had difficulty of breathing, vomiting, and diarrhoea.

I saw her on March 29th, and her condition was as follows:—Anxious expression, bright flush patches on both cheeks. Tongue moist, coated down the centre. Respirations 34. Pulse 120, regular, small volume. Temperature 101.2°. Abdomen much distended all over, tender and tympanitic. Diarrhoea, motions not light coloured. Small swelling felt in left iliac region about size of an orange, indistinct and tender. *Per rectum* ill-defined tender swelling in front and to the left of bowel. Chest: cardiac impulse fourth space behind nipple. Respirations entirely thoracic, the diaphragm did not move at all so far as could be seen. Impaired movement of left base with well-marked dulness behind for about two fingers' breadth. Diminished entry of air into left side of chest. Tympanitic resonance right up to cardiac impulse in front and in axilla. Friction sounds over left base in front. Some rhonchi in right

lung. Frequent slight cough, often very harassing ; some slight mucous sputa. Urine 1024 ; slight cloud of albumen. The first time I saw her I was unaware of her history of stomach trouble, and feeling the lump in the pelvis and the general tympanitis, I diagnosed pelvic peritonitis. Belladonna fomentations and morphia internally were prescribed, which relieved the pain and distension considerably. The temperature and other symptoms continued much the same, the cough troubled her a good deal, but the abdominal pain and swelling became considerably less. On April 8th, about 8.30 a.m., she was seized with severe dyspnoea, and began to expectorate large quantities of gruel-like, highly offensive, purulent matter. The distress of breathing became very great, and she gradually became cyanosed and died of respiratory failure about three hours later.

At the autopsy, made the same day, a very large subphrenic abscess was found arising from a gastric ulcer, a certain amount of general peritonitis, and a large abscess in the pelvis to the left side, its walls formed of matted intestine and uterus and broad ligament.

The specimen which you see consists of the subphrenic abscess with the various organs which formed part of its walls.

On looking at the inner surface of the stomach you see a large oval ulcer, measuring one inch long by half an inch broad, situated on the lesser curvature and posterior wall of the stomach about midway between the cardiac and pyloric ends, perhaps rather nearer the former. It is just at the point where the lesser curvature comes into relation with the lower anterior border of the left lobe of the liver. Close to this, on the posterior wall of the stomach, there are one or two well-marked white scars, where old ulcers have existed and healed. The patient, you will remember, had suffered from dyspepsia for three years.

To return to the existing ulcer, it has formed a punched-out hole in the stomach wall. The lower and posterior portion has adhered to the pancreas. The adhesions, as you see, were not very firm, and gave way in taking out the specimen, so that the ulcer appears to have no base, but during life the base of this part was the pancreas. The other portion, situated on the lesser curvature, has proved much more dangerous ; it has first of all set up a localised peritoneal inflammation with protective

adhesions to the under surface of the left lobe of the liver ; but the process of ulceration has proceeded until the whole thickness of the stomach wall has been destroyed, with the result that portions of the contents of the stomach have escaped through this between the under surface of the liver and the upper surface of the stomach. These irritating matters have set up inflammation, going on to suppuration, and an abscess has thus been begun. Now you may well ask why in this portion the gastric contents should have escaped and set up inflammation, whilst in the other, which ulcerated through to the pancreas, nothing of the kind seems to have happened. The answer is, I think, that the anatomical arrangement of the two parts is different. The posterior part of the ulcer came down directly on to the closely applied surface of the pancreas, a fixed organ, and the adhesions to this surface were sufficient to prevent any escape of contents, but the upper portion was applied to the edge of an organ which moves with every diaphragmatic contraction, so that, what with peristaltic stomach contractions and respiratory liver movements, there is much more likelihood of the adhesions being less completely defensive.

The abscess having once started, the inflammatory process has spread along the lines of least resistance, and it has at length formed this very large many-pocketed abscess cavity which you see. You can pass your finger through the perforated ulcer, and you will find that it runs up vertically behind the left lobe of the liver for about three inches, and that you cannot get further owing to adhesions between the liver and the cardiac end of the stomach. The rest of the abscess cavity, however, surrounds the front and sides of the cardiac end of the stomach, turning over the front and upper surface of the left lobe of the liver until it is stopped in the one case by the falciform ligament, and in the other by firm adhesions between the front of the liver and the front of the diaphragm. At its left extremity it surrounds the upper portion of the spleen, being again checked by adhesions between the spleen and the diaphragm. The spleen is considerably enlarged, being about double its natural size, and is bent round and flattened against the diaphragm in a curious manner. Why this enlargement should have occurred I do not know. The middle portion of the front of the abscess cavity I have not yet alluded to, but its

boundary wall is very interesting. It is composed of adhesions between the anterior surface of the stomach, a large part of the left side of the apron formed by the great omentum, the transverse colon, and the anterior portion of the under surface of the diaphragm. The effect of this tucking up of the omentum and involvement of the transverse colon is that the transverse mesocolon is stretched tightly under the spleen and fundus of the stomach. This tucking up of the omentum is interesting in connection with a paper which has recently been published by Professor Adami* on the function of that body, in which he remarks on the curious way in which it seems to find its way to any focus of inflammation in the peritoneal cavity, and there assist to form adhesions, and so shut off the rest of the peritoneum; it is also worthy of notice in this connection that whilst the left edge of the omentum has thus got itself implicated in the subphrenic abscess, the rest of it had involved itself in the pelvic abscess. To return to the abscess—its surfaces are lined all over with rough, flaky lymph deposit, it contained several ounces of offensive pus, and the diaphragm forming its upper surface under the left lung had become much atrophied, and had ruptured shortly before death. The base of the left lung was compressed, and firmly adherent to the diaphragm by pleuritic adhesions. The left side of the diaphragm was arched upwards considerably higher than the right, evidenced during life by the cardiac impulse being a whole intercostal space higher than normal.

The large abscess cavity forms an excellent example of the subdiaphragmatic or subphrenic abscess secondary to a gastric ulcer; it is sometimes spoken of as a perigastric abscess, and such a term applies particularly well to these cases, for the abscess cavity surrounds the cardiac end of the stomach completely except at the back. It is evidently pathologically continuous with the fistulous tract which leads up behind the liver from the upper part of the gastric ulcer, although, as I said before, the actual anatomical connection was at the time of death, and probably for some time before that, severed by adhesions. Before proceeding to remark on the diagnosis and treatment of perigastric abscess, I should like to try and fit together what was found at the autopsy of this case with the history and symptoms during life.

* 'Canadian Practitioner,' March, 1898.

History and Symptoms during Life.

When thirteen years old, pain on micturition and inflammation of bladder.

For three years indigestion, pain, vomiting, no hæmatemesis.

For some months has been worse and under treatment.

March, 1898, second week. Fell downstairs; hurt left side, and began pain in pelvis.

March 25th.—Acute pain in left side; constipation.

March 28th.—Dyspnœa.

Vomiting.
Tymanites.

Pyrexia.

April 8th.—Expectoration of pus.

Autopsy showed

Old adhesions of omentum to bladder.

Scars of previous ulcers of stomach, recent perforated ulcer.

Gradual formation of large perigastric abscess.

Probably ruptured some adhesions and got general peritoneum infected; starting point of pelvic abscess.

Rapid increase in pelvic abscess.

Impaired thoracic movements from pressure on diaphragm and distended intestines.

From peritonitis.
From distended intestines and stomach due to peritonitis.

From septic absorption from abscesses.

Rupture of left diaphragm, atrophied by constant pressure and firmly adherent above to left lung.

Some such a series of events as this seems reasonable to believe occurred in this case, although the actual commencement of some of the events remains doubtful. Thus there is no evidence pointing to the actual time when the perigastric abscess commenced. Its thickly-coated walls and the appearance of its inner surface generally show clearly that it was of old standing, and probably its formation had been so gradual that it had been merely looked upon by the patient as part of her chronic indigestion. The pelvic abscess, on the other hand, seemed much more recent, and as the generative organs were perfectly healthy, it seems probable that it was secondary to the other abscess.

The diagnosis of a perigastric abscess before it ruptures presents many difficulties owing to its situation, and it is not surprising that so many cases are overlooked or mistaken for something else.

The chief features of these two cases may be stated as under:

(1) Young woman.

(2) History of indigestion lasting over a long

period, with much pain and vomiting; in one case hæmatemesis.

(3) Fulness of epigastric and left subdiaphragmatic region.

(4) Displacement of cardiac impulse upwards and outwards.

(5) Impaired movement of left base.

(6) Dulness and diminished entrance of air over left base behind for two or three fingers' breadth only. In one case some friction sounds.

(7) Impaired percussion or well-marked tympanitic resonance over left front below cardiac impulse.

(8) Pyrexia.

(9) Pain.

As regards the character of the percussion note in front below the cardiac impulse (7); this will depend on many circumstances. If the abscess communicates directly with the stomach it will contain gas as well as pus, hence there will be resonance in front. If it is shut off from the stomach it may be dull; but in my second case, although, as you see, it was shut off from the stomach, it was tympanitic in front. That was, I think, explained at the autopsy. The distended cardiac end of the stomach ran right through the abscess; and not only that, the distended transverse colon was involved and tied up between the abscess and the lower costal margin, so that the impaired resonance which the layer of pus lying immediately behind the thoracic wall might have been expected to produce, was overshadowed by the tympanitic note of the stomach behind and the colon below.

As regards treatment of a perigastric abscess, such as in these two cases, no definite rules can be laid down for all cases. In the first place it is evident that you may determine to adopt an expectant treatment, or, on the other hand, you may adopt active treatment and open the abscess at once. The latter is theoretically the proper course to adopt, for by waiting you are exposing the patient to dangers of rupture into the lung or elsewhere, and the prolongation of septic intoxication, with its accompanying fever. But it is one thing to say open the abscess and drain, and quite another to do it. The operation may be much more prolonged and difficult than you would expect, and the patient is often quite unfit to undergo a prolonged operation. The abscess cavity has many pouches, and is lying amidst and around many viscera, which,

together with the overlying ribs, make it difficult to get at. Even if the abscess is successfully opened and drained, the danger of the case is by no means over. To the physician such cases as these should be a reminder to insist upon prolonged rest in all cases of suspected gastric ulcer until all symptoms have completely disappeared, for then it may be possible to prevent such a complication as perigastric abscess. When it has occurred medicine is practically useless as a curative agent, and surgery is by no means always successful.

NOTES, &c.

The Normal Gastric Movements.—Notwithstanding the very numerous studies which have been made during the past ten years upon the processes of gastric digestion and upon the movements of the stomach, there yet remain large open spaces in our knowledge of the two physiological processes which we have named. The fact that the stomach is an internal organ which cannot readily be observed in its physiological activity has forced us to rely upon results obtained by the use of the gastric tube, or by the passage of œsophageal and gastric bougies, which have given us indefinite information; and yet, as a matter of fact, physiologists have from the earliest times been interested in the two questions of gastric digestion and gastric movements. With the employment of the Röntgen rays in medicine new possibilities for the study of the action of internal organs have presented themselves, and some of the most useful results so far obtained in clinical medicine and physiology, from the use of these rays, are to be found in a paper contributed by Dr. W. B. Cannon in the 'American Journal of Physiology.'

His experiments consisted in administering to cats food which had been mixed with subnitrate of bismuth, which latter substance is impervious to the Röntgen rays, and, by giving a sufficient quantity of food to practically fill the stomach, changes in the contour of this viscus were of course represented with the changes in the shape of the bismuth-laden food mass. A large number of animals were observed over a considerable period of time, and Cannon finds that the stomach

consists, physiologically, of two distinct parts, the pyloric part and the fundus; and that over the pyloric portion, when food is present, constriction waves continually course towards the pylorus. On the other hand, the fundus is an active reservoir for the food, and when salivary digestion is advanced this reservoir squeezes its contents out into the pyloric area. For a thorough understanding of his paper it is necessary for us to remember that he makes the following divisions of the stomach, namely, the cardiac portion, which extends from the left edge of the stomach half across its length, and the pyloric portion, which extends from this point to the beginning of the small intestine. This pyloric portion is, however, divided into two parts—the antrum, which is just at the beginning of the small intestine, and the pre-antral portion, which extends from the middle of the stomach nearly to the pyloric opening.

Cannon finds that the stomach is emptied of food by the formation, between the fundus and the antrum, of a tube along which constriction waves pass; that the food in the fundus is pressed into this tube, and the tube and antrum slowly cleared of food, by means of constriction waves. In other words, the pyloric half of the stomach, called the pre-antral portion, forms itself into a tube which conveys food from the fundus to the pyloric orifice. But the process of propulsion is not all from the fundus towards the pylorus. On the contrary, the food having passed through the tube in the pre-antral portion is once more pressed upon in such a way that the food returns backward along the path which it has followed, and thus becomes thoroughly mixed with gastric juice, so that while its progress is ultimately towards the pylorus, this progress is for a time oscillating in character.

Again, he finds that the pylorus does not open at the approach of every wave bearing food, but only at irregular intervals, and, still more interesting, that the arrival of a hard morsel causes the sphincter to open less frequently than normal, and thereby the passage of the already liquefied food is delayed, for the solid food remains in the antrum to be rubbed by the constrictions until triturated into small particles, which are softened by the gastric juice. If, however, the morsel is too hard to be softened and digested in the stomach, it ultimately escapes into the intestine. Of still

greater interest in connection with these studies are the observations which Cannon has made concerning the function of the fundus of the stomach. He finds that food in this portion of the viscus is not moved by peristalsis and is not mixed with gastric juice, but that the fundus forms a sort of pouch in which salivary digestion of the starches and general softening and maceration of the food can be carried on for a considerable period of time without being stopped by the acid gastric juice. This result is of interest, as it explains the clinical fact that both taka-diastase and pancreatin are known to carry on their digestive function in the stomach for a considerable period of time after food is taken, although theoretically the acid gastric juice should inhibit the activity of the pancreatin at least. It also explains an interesting fact which the writer of this editorial has noticed in a number of cases, namely, that the stomach seems to possess the ability of retaining certain articles and of passing others into the small intestine. Thus if a purgative is taken after food has been swallowed and a copious movement of the bowels is produced within a few hours—as it is, for example, after the use of a saline purgative—none of the undigested food, at least in the normal individual, escapes from the bowel, whereas mucus and other materials are frequently washed out. In other words, it has seemed to the writer that the alimentary canal possesses a selective ability whereby it retains substances with which it has not finished its physiologic function, and passes on those which do not need to be retained in the stomach.

We are wont to hear a great deal at this time of nervous dyspepsia, and undoubtedly nervous dyspepsia arises from the fact that so much nervous energy is expended in the ordinary pursuits of life that a sufficient amount is not reserved for the processes of digestion, and as a result digestion is impaired or entirely prevented. Cannon's studies indicate the fact that marked nervous excitement also inhibits the movements of the stomach. Thus Cannon found, when the cats upon which he experimented became irritable or showed any signs of distress, the digestive movements of the stomach were at once stopped; but as soon as the animals were soothed or their anger appeased, the digestive movements already described at once reasserted themselves.

It is studies such as these of Dr. Cannon which attract the practising physician to the study of physiology, a department of medicine in which many practising physicians are woefully ignorant because they do not understand the direct bearing of physiological knowledge upon the practice of medicine. Much of the so-called physiology taught in the medical schools is theoretical, or so deep as to seem to have little bearing upon bedside experience, and we welcome these studies which, while scientific and accurate in the realm of physiological science, are also productive of increased knowledge which may prove of value to the sick.—*Therapeutic Gazette*, July, 1898.

Cold Sponging v. Cold Bath.—Dr. H. A. HARE states that he has used cold sponging in his hospital practice, and rarely the bath, with the most satisfactory results. His plan is as follows:—(1) In early typhoid, with constipation or moderate diarrhoea, give a full dose of calomel in divided doses in order to stimulate the liver and antisepticise the bowel with bile. (2) Control the fever when it reaches 102° F. by sponging. The patient being stripped and laid on a rubber sheet or blanket over a sheet, he is to be sponged with water adapted in temperature to his needs, and it is to be remembered that the rapid application of a low temperature is more refreshing than the prolonged application of a higher temperature (Baruch). The chief advantage of the cold sponge lies in the shock and reaction. This is better obtained by the use of ice sponging than by the bath. The patient's surface is always bright red in ice sponging, often blue in the bath, and that the fever is not the chief danger in the case renders the fact that as great a reduction from the sponge is not reached as from the bath, of little importance, except in hyperpyrexia. Shattuck tells us that he has found no marked or constant difference in the antipyretic value of cold sponging at 60° F. for twenty minutes, the cold pack at 60° F. for sixty minutes, or the cold bath at 70° for ten or fifteen minutes. Finally, if this does not bring the temperature down to 100.5° or 101° F. in twenty minutes, resort should be had to the tub. It is essential when the sponging is used that more water be applied to the back than to the trunk of the body, for at the back the great muscles and

thick skin retain the heat as a reservoir, and are not cooled if only the front of the body is sponged. Further, the posterior surfaces are the ones apt to be congested and sore, from the dorsal decubitus, and therefore need the stimulating effect of the bath, as do the kidneys and other deeply situated organs. That this treatment is of value is shown by the marked redness of the skin, the improvement of the circulation and respiration, and the cleared mind. (3) It is advisable not only to use friction in a light form, but to use moderately active massage, with the same objects in view as when the rest cure is undertaken, for the proper treatment of typhoid is a modified rest cure. Dr. Hare believes that by this means bedsores, local congestions and effusions, œdematous swellings, peripheral nerve pains, and muscular feebleness will be largely decreased, and Popischl has shown that mechanical irritation of the skin is capable of increasing heat loss 95 per cent. (4) In nearly all cases give more nourishment than the average typhoid patient in the past has usually had. With the exception of broths and meats, almost any article easy of digestion should be allowed—as one or two or more lightly boiled eggs, corn starch, arrowroot, &c. (5) Use stimulants in carefully graduated doses whenever the circulation needs them, particularly alcohol. Even the cold bath enthusiasts give whisky to overcome the depression they often produce.

The Charlotte Medical Journal, July, 1898.

J. L. MILLER concludes an article on "The Smegma Bacillus" in 'Medicine,' July, 1898, with the following summary:

(1) Over the entire surface of the body and exposed mucous membrane, and especially on the genitalia, bacilli are found which resemble morphologically and in tinctorial qualities the *Bacillus tuberculosis*.

(2) From the external genitalia they frequently gain access to the urine, especially in women, and may be a source of error in the examination of the urine for tubercle bacilli.

(3) The smegma bacillus presents wide variations in size and form, thus rendering morphological differentiation frequently impossible.

(4) While most smegma bacilli are more readily decolourised by any of the solutions commonly

employed, occasionally they possess equal or even greater resistance than the tubercle bacillus.

(5) Methods of decolorisation where acids are employed alone are especially fallacious; acid alcohol or dilute alcohols, unless long continued, are equally unreliable. Better, but not free from error, is the use of absolute alcohol for at least five minutes; in ammoniacal urine, however, such prolonged use of alcohol may also remove the stain from the tubercle bacillus.

(6) Attempts to remove the fat or fatty acids from the bacilli by ether, chloroform, or other solvents fail to furnish us with a means of differentiation.

(7) We must rely on means of excluding the smegma bacillus from the urine. It has never been demonstrated in the bladder, and apparently seldom invades the deep urethra; therefore, by cleansing the external meatus and withdrawing the urine with a catheter we can exclude this possible source of error.

Treatment of Lupus with the Röntgen Ray and Concentrated Sunlight has passed beyond the experimental stage. H. Kümme, of Hamburg, announced at the German Congress of Surgery that he had positively cured sixteen patients to date with exposure to the Röntgen ray for fifteen to twenty minutes, twice a day, for a period of four weeks to several months. The sound skin is protected by a light sheet of lead or stanniol, cut to expose only the lupus patch, with the tube from 20 to 40 cm. distant. All irritation of the skin is most carefully avoided, and exposure individualised to prevent the slightest dermatitis. The lupus patches heal over with a smooth surface scarcely to be distinguished from normal skin. Similarly favourable results have been secured by Finsen, of Copenhagen, with contracted sunlight or electric light, excluding the heat rays. As blood prevents the passage of the rays he expels it from the point to be treated by a convex, transparent glass saucer, strapped on tightly. He reports a large number cured from one to two years, but his process requires months of treatment, as only a small point can be treated at a time. The combination of this method with the Röntgen ray treatment promises remarkable results. The latest tests fail to confirm the bactericidal action of the Röntgen ray. Its effect on

lupus is evidently due to some still undetermined influence on the lupus tissue, possibly electro-chemic or trophoneurotic.—*Cbl. f. Chir.*, July 2nd.

Resection of the Sympathetic.—Messrs. Combemate and Gaudier, of Lille, showed a patient before the Paris Academy of Medicine at the meeting of April 19th. The patient was the subject of exophthalmic goitre, and she had been operated on by resection of the sympathetic. The results were as follows: first, a most important diminution of the exophthalmos; secondly, lowering of the heart beats from 200 to 100 in the course of a week, while at the same time the painful pulsation in the præcordium disappeared; and thirdly, there was no marked effect on the goitre itself. Messrs. Combemate and Gaudier concluded from their observations that in cases of exophthalmic goitre, when the tachycardia is intense and continuous, when this is the only symptom, or when there is also exophthalmos, there is always the risk of dangerous symptoms supervening. In such cases section of the cervical sympathetic, which acts most probably by preventing the hypersecretion of the thyroid gland, is the best operation to do.—*Medical Record*.

A very Delicate Test for Bile Pigment.—By Drs. Krokiewicz and Batko ('Wiener klinische Wochenschrift,' 1898, No. 8; Ref. 'New York Medical Record,' liii, 1898, No. 18, p. 628). The following reagents are necessary:—(a) A 1 per cent. aqueous solution of sulphanilic acid; (b) a 1 per cent. aqueous solution of sodium nitrite; (c) pure concentrated hydrochloric acid. The test may be made according to one of three methods: 1. To two c.c. of the reagents (a) and (b) add from two to five drops of the urine, and shake; if bile pigment is present a ruby-red colour develops, which changes to amethyst violet upon the addition of one or two drops of hydrochloric acid. 2. To a few drops of the solutions (a) and (b) add an equal quantity of urine and one drop of hydrochloric acid, and mix; a deep violet colour results. 3. Shake together in a test-tube several drops of the solutions (a) and (b), and pour out; if five c.c. of icteric urine be added, the mixture turns ruby-red, changing to amethyst violet upon the addition of hydrochloric acid.

Post-Graduate, July, 1898.

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NOTICE.

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ON SOME POINTS RELATING TO APPENDICITIS.

A Clinical Lecture delivered at St. George's Hospital,

BY

WILLIAM H. BENNETT, F.R.C.S., &c.,
Surgeon to the Hospital.

GENTLEMEN,—I have no doubt some of you have heard me say before that I think a much more useful purpose is served in lectures like these by the consideration of diseases which are common, not only in the hospital, but also amongst the community at large, rather than of those which are rare; for the obvious reason that you are more likely to have to deal with those which are common. Appendicitis is a disease which I suppose may fairly be called common, both in the hospital and outside. I propose, therefore, to consider to-day some practical points connected with it.

Before entering upon a discussion of the question, let us be sure about our position with regard to the term; in other words, let us distinctly understand what most of us mean at the present time when we use the term "appendicitis," because in this respect a loose habit of speaking has grown upon us, which is not altogether advantageous. There is a certain train of symptoms, characterised for the most part by a more or less sudden onset of pain, generally although not always accompanied by vomiting, high temperature, and very often by rigors, together with fulness, dulness, and tenderness in the neighbourhood of the right iliac fossa, the tenderness being, in the majority of cases, more or less concentrated in a certain spot known as McBurney's point. Those are the symptoms which are usually described as being indicative of the complaint called appendicitis. Those symptoms are not, however, necessarily due merely to a disease of the appendix; in a good many instances, therefore, it is quite incorrect to describe these cases as cases of appendicitis only. You may take it for practical purposes there are

four distinct classes of case which give rise to these symptoms:—(1) APPENDICITIS, in which the appendix itself is diseased only. This condition may, however, be a tuberculous manifestation. (2) TYPHILITIS, in which the cæcum is the seat of the disease, the appendix being for all practical purposes healthy. Such cases you may find following upon dysentery and typhoid fever. (3) PERITYPHLITIS, in which there is no obvious disease in either the cæcum or the appendix, but in consequence of surrounding inflammation the parts have become bound down by adhesions to a variable extent. (4) COMPOUND CASES, in which the cæcum and the appendix are both diseased at the same time, although it is not always easy to say in which of these the disease originated; the practical point being that the two structures are involved in the disease.

Of these the commonest variety is appendicitis, but the cases in which both appendix and cæcum are involved at the same time are moderately frequent.

Those, then, are the four ordinary conditions which may give rise to the symptoms to which I have referred. There is, I believe, a fifth condition which I venture to call VOLVULUS OF THE APPENDIX. I have not seen it described hitherto. The following cases are typical examples of the conditions mentioned:

APPENDICITIS.—A young man, 25 years of age, was seized suddenly with pain in the right iliac fossa, of the ordinary type occurring in what we call appendicitis. He had a temperature of 105° , some fulness, loss of resonance, and the characteristic tenderness, the attack having been preceded by the eating of half a melon. The case was a good example of the usual symptoms of an acute attack of appendicitis. The first attack subsided, but he subsequently had two other attacks, which led to operation in the ordinary way. Upon opening the belly the appendix was seen lying just inside the peritoneum. It was somewhat bulbous in shape, with a large thick end, but there were no adhesions of any kind. The cæcum was quite free, and the operation for the removal of this isolated appendix, thickened as it was, and having no adhesions, was so simple, and occupied such a short time, that one felt some doubt whether it was right to accept the large fee which is commonly charged for these operations. The disease was

entirely limited to the appendix, which at the point of section was perfectly healthy. On the distal side there was a stenosed part, and beyond that the appendix was thickened and somewhat dilated. The disease was reported after examination to be tubercle. That is a good example of disease confined to the appendix.

The following is an example of disease of the cæcum—TYPHILITIS, as it used to be called. The patient, a man, had been in India many years, and had had both dysentery and typhoid fever. There had been several attacks of discomfort about the right iliac fossa, sometimes associated with diarrhoea, sometimes with constipation. As a rule, perhaps, the diarrhoea was more manifest than the constipation. On two occasions there had been vomiting. These attacks were ascribed to "relapsing appendicitis," as it was called. He was never quite well between the attacks, and so he came home. When the patient came under observation he had had twenty or more attacks of this so-called appendicitis. He was seen in an attack at home, and had the ordinary symptoms I have described. The tenderness was rather diffuse, and not quite so acute as in the majority of the cases I have seen. The vomiting was very severe; the temperature was 103° . The attack subsided with an attack of diarrhoea, which is not at all uncommon in these cases. It was thought right to operate upon him for several reasons, and so I performed the operation. The appendix was practically healthy, but the cæcum, on the other hand, appeared to be to a great extent cicatricial, the result, I suppose, of old dysenteric ulceration. So thin, however, was the viscus at one spot that in separating a slight adhesion to make a proper examination I tore a hole in the gut. The opening was sewn up, and he did well in the ordinary way. Here was an example of a case in which the disease was limited to the cæcum; and, as I said just now, such cases are generally those which occur after typhoid fever or dysentery, as the case may be.

The next is a COMPOUND CASE, the appendix and the cæcum being diseased at the same time. A man about thirty-three years old had the usual train of symptoms, and in this case between the attacks (of which he had had eight or ten) he was always conscious of a certain amount of discomfort about the right iliac fossa. He almost always had constipation, and occasionally he had profuse

attacks of diarrhoea. Whether these latter attacks had anything to do with the disease in question I do not know. At all events, the case was diagnosed in the ordinary way as appendicitis, and I operated upon the man. The appendix was very large, almost the size of my index finger, thick and hard. Everything was very much matted together, and at the end of the appendix there was discovered an abscess of moderate size. On pressing upon the walls of the abscess it could be emptied with perfect ease, a circumstance which is not common in an abscess connected with the appendix. To isolate the appendix it had to be separated from the sac of this abscess. After separating the appendix from the abscess sac there was found to be a hole leading into the cæcum, through which I could pass the tip of my index finger; this accounted for the ease with which the abscess could be emptied by pressure. This was a case in which the appendix was extensively diseased, and the cæcum so much so at the same time that a portion of its wall had disappeared, and the abscess, which on the one hand was associated with the appendix, could be squeezed into the cæcum through this large opening which existed. Where the primary disease had commenced it was impossible to say, but there was no doubt of the two structures being involved in the disease at the same time. The appendix was removed and the opening in the cæcum sewn up; the man did well.

A case showing the effect of a few adhesions about the cæcum, the gut itself and the appendix being apparently free from disease (PERITYPHLITIS) is the following:—A man forty-three years old was subject to occasional attacks of constipation, which, if they were at all exaggerated, always terminated in pain and tenderness in the right iliac region, accompanied by vomiting and fever. The attacks usually passed off, when managed in the ordinary way, in the course of three or four days; but on one occasion an attack lasted ten days. There was nothing to distinguish the case from one of relapsing appendicitis. I therefore operated, and found the appendix normal; the cæcum appeared also natural, with the exception of some rather firm adhesions binding it to the wall of the iliac fossa. On separating the small mass of adhesions a caseated flattish nodule was found in its centre. The cæcum was freed thoroughly, the man

recovered as usual, and when last heard of had had no further attacks since the operation.

There is another class of case to which I wish particularly to call your attention in which a patient suffers from attacks of what are indistinguishable from what we call appendicitis; the pain and tenderness are the same, and there are all the symptoms which we associate with that complaint, excepting perhaps that the subsidence of the symptoms is rather rapid. And yet, when the surgeon cuts into the belly in the quiescent stage, he finds an apparently healthy condition of parts. These when not purely neurotic are, I believe, cases of VOLVULUS OF THE APPENDIX. The following is an example:

I operated upon a girl about five years ago, who I was assured had had three or four attacks of typical appendicitis, and from the description there was no doubt she had so suffered. I operated on the strength of this history. It is true I could feel nothing at the time of my examination of the patient; there was no tenderness, but that is also often the case in what is commonly called relapsing appendicitis. On opening the belly, as far as I could see everything was healthy. The only thing I noticed to be a little peculiar was that the appendix seemed to be slightly constricted at a point about three-quarters of an inch from the cæcum, but not more so than I have seen many of these appendices in the post-mortem room, and I should not have thought anything of it had not this girl had such very characteristic symptoms. Under these circumstances the question arose what was to be done? As far as I could see I had cut into a healthy belly in a patient who had had symptoms of appendicitis three or four times. There was nothing neurotic about the aspect of the case; I therefore assumed that there must have been some cause for the attacks, and so, as there was this little depression in the appendix I thought I would remove it. I did so, and upon opening the appendix after removal this depression was found to be of no importance; there was no organic constriction or anything of that kind, and the appendix seemed to me to be quite healthy, except that perhaps *it was a little more flabby than usual, as if it might have been abnormally dilated at some time.*

The patient, of course, did well after the operation, and she has never had another attack of the

symptoms. I was very unhappy about this case, because I have a prejudice against doing operations when they are not necessary, and I could not help feeling for some time that I had in this case done an operation which was really uncalled for. It is true, however, that the symptoms never recurred, and I have no doubt now, viewed by the light of a case which I had the other day, that I was quite right in doing as I did in this girl's case, and that the symptoms from which she suffered from time to time were due to a volvulus of the appendix.

A patient a few months ago consulted me with regard to symptoms which appeared to be those of relapsing appendicitis; an operation was therefore advised, and I carried it out. On opening the belly there appeared to be some old scars about the cæcum, otherwise everything seemed quite healthy excepting a small soft adhesion which tied the cæcum slightly to the abdominal wall. The appendix was very much of the normal size, but it was pointing rather forward into the belly; and whilst we were looking at it, the appendix, quite straight and normal so far as we could see, gradually following apparently upon some peristalsis in the cæcum, began to stand a little erect, and then falling over it bent on itself. In point of fact it was a good example of what may fairly be called volvulus of the appendix. It appeared as if the bending over of the appendix in this way was due to the fact that its little mesentery was rather short, and so was abnormally dragged upon. When the cæcum began to contract in a certain direction it seemed to pull the appendix so that it got twisted, just as occurs in twisting or "volvulus" of the small intestine, for example. There is no doubt that this appendix, judging from the look of it after removal, had been from time to time greatly distended. As far as I can explain the case it seemed to me that this peculiar change occasionally took place, and that in consequence of the twisting of the appendix upon itself in this way it must have become distended, and so given rise to the symptoms of appendicitis. Later, I suppose, some other little movement occurred before peritoneal adhesion could take place, and the small piece of gut unwinding itself was emptied, and so the symptoms subsided. I do not know that any other explanation will fit the circumstances. The explanation in the girl's case which I have

mentioned must, I fancy, be as follows:—She occasionally had these twists of the appendix, which, after it had become distended to a certain extent, were rectified by the uncoiling of this little piece of gut. Of course the removal of the appendix in such a case would naturally cure the patient. In the other case also I removed the appendix. Although the cæcum was not in a very healthy condition I did not interfere with it, because I did not see any reason for supposing that it was the cause of the trouble. All I did was to remove the appendix and separate a small adhesion which seemed to tie the cæcum to the abdomen, and so prevent its free action during peristalsis. I have not seen any mention of cases of volvulus of the appendix in the ordinary surgical literature.

Taken together these cases which I have described are good examples of what we speak of sometimes very inaccurately as appendicitis. As you know, in these times there is practically only one treatment for appendicitis, unless the symptoms entirely disappear after the first attack. If a patient has what is called a "relapsing appendicitis," with most of us there is only one treatment, namely, operation. Removal of the appendix is usually recommended without hesitation if a second attack occurs; it is then considered almost as a matter of course that the patient's interest will be best consulted by the removal of the appendix. Practitioners of some few years ago used to see as many cases of appendicitis as we meet with now. But the operation for the removal of the appendix at the time I am speaking of was practically unknown; that is to say, twenty or twenty-five years ago. Now the experience of practitioners of that time, although I fear some of you are inclined to think them old-fashioned, is extremely valuable, and their experience undoubtedly goes to show that a number of these cases of so-called appendicitis do not end disastrously if they are not submitted to operation, and that some cases in fact get perfectly well after a series of attacks. It is not long ago that a practitioner of large experience asked me what the justification really is for operating to the extent we now do upon these cases, seeing that in his experience a considerable number of cases, if left alone, certainly do not die, and sometimes get perfectly well. A question like this is not always easy to answer

at once. Of course there is not the least doubt that many of the cases would not die of the disease if left alone, and that a certain proportion of people suffering from relapsing appendicitis recover completely. I think I know now at least a dozen people each of whom has an attack of appendicitis from time to time. The patient lies up for a few days during the attack, then seems to get quite right until the next attack, and excepting for the inconvenience he does not seem any the worse. They are all hale men, and not one of them has the least intention of being operated upon. I do not think that the life of any of these people is likely to be shortened at all by their declining to submit to operation. If, then, it is a fact—and I have no doubt it is a fact—that some of those who suffer from this so-called appendicitis do not risk their lives in these repeated attacks, why are we so very anxious to operate in all cases? What is the justification for our position in the matter? That is the question some of the older school of practitioners ask. The justification is this:—We know quite well that although, as I have said, a certain percentage of these people would not die of these attacks, a considerable percentage of cases would prove fatal sooner or later, supposing the diseased appendix is not removed. *At the same time we have no means in the absence of obvious signs of suppuration, as far as I know, of judging in the least whether any given case is one of those likely to be associated with great risk or not.* We cannot, in fact, tell which are the cases that are dangerous to life as distinguished from those which are not; and as we do not regard the operation now with any great degree of anxiety, so far as its immediate risk is concerned, it does seem on the whole safer to advise operation as a rule in order to be sure, as far as one can be sure, of saving the lives of those who happen to have the type of the disease which would be likely to prove fatal. That is the justification for our present line of treatment. It may not be a very strong justification at first sight, perhaps, but a very little consideration will show you that it is sufficient.

It has been said that chronic cases and those which are insidious in their onset, giving rise to subacute symptoms only, might be with safety ignored so far as operation is concerned. But that is entirely wrong. It was also said that the

cases which in their original onset were very acute were the most commonly associated later on in the relapsing period with the greatest risk to life. That again is not always the case. For example, I operated on a case only the day before yesterday, the patient being a man who had had practically no symptoms worth mentioning excepting constipation and a lump in the right iliac region, with very slight tenderness indeed. He had had three attacks, and between the attacks he never seemed to be quite well. He was a professional man in active practice, and he thought he could get along well enough as he was, provided the attacks did not become more frequent. As he was not a very strong man in some respects, and as other matters pointed to the desirability of his being relieved of the disease, I thought it better for him not to run the risk which these repeated attacks involved, and therefore I operated. Now, although he had had so few symptoms, and had suffered so little, it was one of the most difficult cases of the sort that I have had to deal with. It took us one hour and a half to remove the appendix, and we found running behind it an abscess about two inches in length, full of the most poisonous material; the abscess was so situated that it travelled towards the general peritoneal cavity, into which there is no doubt that before very long it must have burst, probably with fatal effects. The case is a good example to show how impossible it is to judge of the character of these cases by the mildness of the symptoms or the insidious way in which the disease comes on—a most important point to bear in mind. On the other hand, I have operated on cases in which the onset has been most acute, so that if there were any relation between the acuteness of onset and the risk to the patient's life, it must have existed in such cases. Yet I have found the appendix in some of these very slightly diseased, in fact merely a little adherent. I do not know of any more important clinical fact connected with the subject of relapsing appendicitis than the entire inability of forming from the mildness or severity of the symptoms any reliable estimate of the gravity of the local conditions or of the difficulties of the operation requisite for properly dealing with them.

ON PRACTICAL POINTS IN PERCUSSION AND AUSCULTATION, AND IN THE PHYSICAL EXAMINATION OF THE CHEST.

ABSTRACT OF LECTURES TO ADVANCED CLINICAL STUDENTS

BY

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III.

ON THE PRACTICAL EXAMINATION OF PATIENTS, AND ON THE "SPECIAL" PERCUSSION AREAS OF THE CHEST.

A thorough physical examination of the chest is an exceedingly long and laborious undertaking, even for the trained observer. You will learn by experience when you have been in practice how to economise as much as possible your time and labour; but I may at once give you a few hints in that direction.

Two broad cases arise. The patient presenting himself for examination may be to all appearances healthy, or he may not. In the first case a general exploration will suffice. In the second this will only be preliminary to the "searching" examination.

The General Exploration.

In the manifestly healthy, as in most persons applying for certificates of soundness, it is generally easy to exclude phthisis. The eye will help you both as regards the general aspect of the patient, and the configuration and movements of the chest. Where the movements appear to you to be deficient, palpation may decide the point, but the quickest method is that of "resisted respiration," which I have previously described, and which is specially applicable to the pectoral and to the lateral regions of the chest. Firm pressure of the hands is made on opposite surfaces of the chest, whilst the patient is directed to breathe deeply. The effort thus induced brings out a relatively full inspiratory excursion, and the expiratory recession is aided

by the pressure; in this way the respiratory range can be got at.

One advantage of this method is that it may be applied without removing the clothes; but it is far more reliable if the chest be stripped, when any suggestions arising from the conformation of the thorax, from its lack of symmetry or of mobility, &c., are at once perceived.

I have already dwelt at some length upon inspection and palpation because they teach us much, and are the most rapid and the least trying of all our methods of examination. I have but a few remarks to add in connection with inspection.

There is a general tendency even among senior clinical pupils to limit their inspection to the front of the thorax, as though, like the moving shadows in Plato's cave, the patients possessed only two dimensions. The chest being a cube, not a surface, we cannot form a complete idea of it until we have looked at it from all sides. We must either walk round the patient, or cause him to turn in front of us.

Passing now to percussion, in the cases in question a rapid regional examination will usually suffice to enable you to pronounce the chest healthy. First ascertain that the patient has his heart in the right place by feeling the apex-beat. Percussion is then to be applied to the cardiac area and to the hepatic area to detect the absence of emphysema, to the infra-clavicular and supra-spinous regions to eliminate phthisis, to the anterior and lateral bases to exclude the possibility of effusion or of pleuritic thickening, to the posterior bases with the same object, and also with that of identifying the normal extent of the pulmonary expansion, and lastly to the mid-dorsal region as a matter of course.

By degrees you will succeed in doing all this rapidly, for this regional exploration is best performed with the "single stroke" and "free-hand" percussion, that is with a swinging blow from the elbow and the wrist (never from the shoulder), and with a firm pressure of the finger which receives the stroke, the object being to elicit the resonance of a considerable surface and depth of lung. The satisfactory conclusion arrived at must, however, in some cases be checked by a more careful percussion, and subsequently by auscultation at the apices, as, for instance, in all delicate

young people, and in subjects otherwise liable to suspicion. In all others you will probably feel quite justified in signing after a carefully performed general exploration such as I have described.

The Searching Examination.

Where disease is either apparent or suspected, greater attention is needed at each stage of the examination; although for the remainder of the chest, in affections of limited extent, such as an apical phthisis, a general exploration will suffice. It is here that ample time is indispensable; but it will be best economised by *not being in a hurry to percuss*. Trust largely to inspection and to palpation for broad conclusions. Important changes never fail to reveal themselves to the eye and to the touch. You may be able to identify large effusions, pneumothorax, chronic pleurisy or pulmonary fibrosis, emphysema, and even phthisis, without a stroke of percussion. The aspect and shape, the respiratory mobility or inertia, the elastic yielding, or the resistance of the thorax, will teach you more and more as you attach more importance to their investigation.

In your inspection take a careful sight, either horizontally by bending to the level of a recumbent patient, or from the side, from above, or from below if the patient be standing or sitting. The slightest unevenness of movement can then hardly escape your notice.

Bimanual palpation, one hand making pressure upon the upper pectoral, the other upon the upper scapular region, will give you quickly definite evidence as to any want of respiratory activity at the upper part of the right or of the left chest, and at the same time as to their elasticity; and the same method is to be applied from side to side at the lower axillary levels.

Above all localise at once the apex of the heart with hand or stethoscope, as a rapid though indirect help to diagnosis. Considerable displacement being often due to pulmonary or pleural causes, important conclusions may be drawn without any loss of time as to their presence or probable absence.

Not until these diagnostic resources have been exhausted need percussion be applied. Its performance is a repetition of that which has already been described. You have localised the region of the disease, and you must now localise its

extent. This involves the finest percussion which a firm pressure by the percussed finger combined with extreme lightness of stroke by the percussing finger can provide. But you may obtain more decisive and rapid results if trained to the use of the pleximeter. The limits of the dulness must be traced, not on one side only, but as far as possible on all sides. It is in this finer work that inherent thoracic resonances and dulnesses must be taken into account, and in this connection a few remarks are necessary.

THE THORACIC SKELETON AND ITS PLEXIMETRIC BONES AS INFLUENCING PERCUSSION; AND THE AREAS FOR PERCUSSION PRESENTING PECULIARITIES AT THE FRONT OF THE CHEST.

We now come to the special application of those general principles of which my first lecture was intended to give you an elementary idea. Throughout our examination of the chest we have constantly to bear in mind the local peculiarities which arise from the thoracic conduction of vibrations, and from the consonating or damping influence of neighbouring viscera.

It is most important that we should not mistake for disease those local peculiarities which really belong to health. With some of these you are familiar in front of the chest; for instance, the cardiac dulness on the left side, and that of the liver on the right, which establish the chief difference between the normal percussion results in the two halves of the thorax. If everything were symmetrical, percussion would be deprived of some of its difficulties. An accurate knowledge of the normal disparities is therefore essential to an advanced percussor.

But in addition to the non-symmetrical areas we must note those regions where, for anatomical reasons, the percussion note is of a different character from that which we might expect, and does not conform with the standard of resonance which at other parts of the chest is the normal standard. These areas are—

- The supra- and infra-clavicular,
- The axillary,
- The manubrial and upper parasternal,
- The lower sternal, and
- The anterior basic regions.

Having already dwelt upon the special features

of clavicular and sternal percussion, we need only consider the first three regions in this list.

The percussion of the apex and its uncertainties.—

The supra-clavicular and the infra-clavicular fossæ, but chiefly the former, as well as the supra-spinous fossa, often tax the judgment even of experienced percussors. There is so much bone and so little lung.

The sterno-clavicular hinge allows the clavicle a large angle of movement, and as the shoulder rises the supra-clavicular fossa deepens, and the lung is more out of reach. Quite apart from disease, the shape of the chest, and the slope of the clavicle and of the ribs vary considerably in different subjects. But in emphysema and in asthma the ribs themselves and the sternum are raised to the utmost by tonic muscular effort, and the fossa becomes considerably broadened as well as deepened, whilst in the drooping chest of advanced phthisis it may be so narrow and hollow as to render percussion difficult.

In a proportion of cases nature conforms to the teaching of books, and in them we may find the pulmonary apex rising above the level of the clavicle; much more often it does not. The relative level of the apex varies in every case.

In all this there is therefore much uncertainty. But this is not all. Normally the lung should rise above the anterior level of the first rib; but here also variability prevails. And again I need only allude in passing to swollen glands or veins, and to other abnormal contents which may complicate the percussion of this fossa; but their nature is usually obvious.

Moreover, it is difficult to know what the finger is percussing at the bottom of the hollow, whether lung or bone. I think that the supra-clavicular percussion note is largely pleximetric; and you will notice the amount of surface presented by the flat of the first rib and by the transverse process of the seventh cervical vertebra when you view the fossa from above in the skeleton. In percussion the left finger probably always rests in part upon one or both of these bony surfaces.

Fortunately the first rib is a faithful pleximeter, and the dulness from any induration of the apex will be the more readily transmitted by it as close adhesions are apt to be set up by apex disease. Dulness would therefore imply thickening of the lung or pleura.

There is a disturbing element, however, which may arise from pleximetric conduction. A resonance may be conveyed from the trachea which may veil any slight pulmonary dulness. This risk renders the direct percussion of the narrow thoracic outlet at the inner corner of the fossa less conclusive than a percussion of the outer portion of the fossa where there is no lung. The note which is there obtained is a conducted note it is true, but it is a note conducted from the lung only, and not from the trachea. You will bear in mind, therefore, that a "boxy" note obtained at the inner side of the supra-clavicular fossa does not inevitably mean a vomica. More often it is merely the transmitted tracheal note.

In conclusion, although so important for diagnosis this region is disappointing as a field for percussion, and it also presents difficulties in auscultation. In the first place it is not uncommon for the supra-clavicular percussion to be duller on the side where the better note was obtained by direct clavicular percussion. Again, there is a danger of attributing a boxy percussion note to the underlying lung whilst it may be due to conduction from the neighbouring trachea, or the percussor may over-rate the size of the surface of lung rising above the clavicle; often enough the lung is entirely below its level. For a satisfactory percussion the pleximetric finger must be applied firmly to the bottom of the fossa, and if possible inclined away from the trachea, and light percussion applied as well as stronger percussion. With these precautions percussion of the fossa is more reliable than that of the inner half of the clavicle itself.

The infra-clavicular fossa.—Similar considerations belong also to the percussion of the infra-clavicular fossa or first interspace, a great portion of which is seen in the skeleton to be a bony surface. It presents no mechanical difficulties except the depth at which the lung is often contained, and the looseness of the intervening soft parts. The finger has to be pressed horizontally against the first rib or the first interspace, and not allowed to span two ribs. Very careful exploration is needed at the sternal end of the first interspace. slight dulnesses being easily disguised by the loud tracheal resonance which a heavy stroke would elicit. Here, again, the outer district yields more reliable results than the inner one.

The upper axillary region is an important area by reason of its relative distance from the root of the lung. Its percussion may often yield evidence of apex disease which had been overlooked anteriorly. If the patient's arm be fully raised the finger can reach high up and percussion can be applied to the pulmonary surface with the intervention only of a thin covering. The same advantages render it an important area for auscultation. In respect of its relative freedom from conducted tracheal and bronchial resonance it contrasts with the supra-clavicular and the supra-spinous fossæ.

DORSAL PERCUSSION AND ITS "SPECIAL" AREAS.

Unlike the front of the chest, of which the outward features and the underlying organs are fairly familiar, the back of the chest for many of us is, like an unexplored continent, a blank. A map of it is wanted which will remind us that it is not a purely pulmonary space, and I propose to suggest to you the outline of that rough sketch.

The advances recently made in clinical teaching are such that I have no hesitation in bringing before you now subjects which a few years ago I could only mention with some diffidence lest they should have been regarded as of doubtful accuracy and of little practical use. I think many of you will now be able to follow me in tracing out the organs in the back by the well-defined though moderate alteration which they occasion in the general resonance of that region. I warn you that, owing to the force which is necessary, the percussor's finger is apt to suffer unless skilfully used, and that Sansom's pleximeter greatly facilitates the work.

The lower limit of the adult lung is, in health, the twelfth rib, to which the diaphragm is attached. In disease, and after death, it may be pushed up one or two spaces. But exception must be taken to the current statement that the lower fringe of the lung normally corresponds to the tenth rib. This can be disproved by percussion as well as by auscultation. In any sound adult the note should be resonant down to the twelfth rib. This fact is of practical importance, but it is also essential to the appreciation of the points to be described.

The posterior thoracic surface presents few external landmarks, practically speaking only the spinous processes and the scapulæ. The borders

and angles of the latter can be made out by palpation. Palpation also will inform you as to the position of the ribs, and particularly of the eleventh and twelfth ribs, which are important as boundaries.

How to find the eleventh and twelfth ribs.—A simple test does away with all difficulty in identifying these two ribs. Feel for their free extremity. The tip of the twelfth rib will invariably be felt in the back; that of the eleventh can only be felt from the front.

The upper limit of the lung in the back is on a level with the first and second dorsal spines. Both apices being narrow (but the left specially so), they correspond only to the inner third of the length of the shoulder. The resonance of this inner third is intensified by that of the trachea, which is transmitted through the heavy vertebral masses and through their long transverse processes. Outwards the resonance is much modified by the thick muscles of the shoulder, and can only be elicited by powerful pleximetric pressure and a heavy stroke. Nevertheless this region may yield important comparative results; but it should be noted that the tip of the acromion and of the clavicle give us a more resonant note than the soft parts, although further removed from the lung.

In the back the surface immediately underlying the parietes is that of the lung, and of the lung only. For the percussor, however, this simplicity disguises considerable variety, which comes out in percussion, and the details of which are included in our rough diagram of the organic districts.

The following are the special regions in question:

- (1) The spinous processes and
- (2) The vertebræ,
- (3) The scapulæ,
- (4) The interscapular dull area,
- (5) The cardiac dulness,
- (6) The splenic dulness, and
- (7) The hepatic dulness.

This leaves very little at the surface of the back, which is not a special region; indeed, the supraspinous fossa also is specialised by its heavy muscular coverings and by the small amount of lung which it contains; and the peculiarities of the scapular region have been sufficiently insisted upon in my first lecture. The really uncomplicated areas are the lateral regions of the back.

The supra-spinous fossa.—The resonance of this region is almost purely pleximetric. At its inner extremity the transverse processes extend far outwards, and the upper ribs reach relatively high, covering the only presenting surface of the lung, which is already out of touch when the middle of the length of the shoulder is reached. Any resonance further out is purely a conducted resonance, and, as I have pointed out, this pleximetric conduction is provided by the scapula and its spine.

As in front, and for the same reasons, apex percussion is least ambiguous when performed outwards. As we approach the middle line confusion may come in. Indeed, the tracheal note, against which we have to guard in front, is yet more prominent here. The loudness of the percussion note obtainable up the entire cervical spine will probably astonish you, and as you pass outwards from the seventh spine to the inner corner of this region you will realise that the tympanitic note which in thin, narrow-chested subjects might suggest the existence of a cavity, is often nothing more than tracheal resonance; and this may extend both outwards and downwards over a rather large surface.

The lateral dorsal or post-axillary regions are fully resonant in their upper two thirds, and the *scapular* regions are also, as previously stated, completely resonant.

Dorsal percussion has also to deal with four areas of partial dulness which present rather more difficulty—the interscapular, the cardiac, the splenic, and the hepatic. As senior students you should be trained not to overlook their presence, although hitherto they have obtained little recognition. Now and then, owing to more than customary attention to basic percussion, the posterior splenic or, more often, the posterior hepatic dulness is perceived, and a false alarm is raised. I have known this highly creditable difficulty occur to some of my house physicians and to fellow practitioners. It will be best avoided by always taking notice of these normal dulnesses in our routine percussion. Their individual features will be the subject of some future demonstration.

The interscapular region is of special interest. The spines, as stated, are normally resonant in spite of the mediastinal solids. But the latter impart to this region a partial or modified dulness of definite shape with which it is important that

physicians should be familiar, since a departure from it occurs in diseases of considerable gravity.

The interscapular dulness may be described as diamond-shaped, with a long vertical axis extending from the fourth to the seventh dorsal vertebræ, and a rather shorter horizontal axis. The spinous processes themselves are not dull, and there is on either side of them a narrow strip of modified vertebral resonance. The less pointed lateral angles of this dulness extend about two and a half inches on either side of the middle line almost symmetrically. This width varies in disease, but it seldom is so much increased as to reach beyond the posterior scapular line, though I have known this occur in lymphadenoma. In the absence of mediastinal disease the area of impaired resonance varies in individual subjects, but this normal dulness can always be traced.

The mediastinal structures to which the interscapular dulness is due are the great vessels in front, and particularly the arch of the aorta and its descending portion, the mediastinal fibrous tissues and fat, the lymphatic glands, more specially those beneath the bifurcation of the trachea, and the roots of the lungs, which, in addition to the resonant bronchus, contain blood-vessels and various solid structures. The left and the right auricle also contribute slightly to this dulness from below.

The cardiac dulness is situated immediately below the interscapular, and between the two there is no strict boundary. It is comprised between the levels of the seventh and of the tenth dorsal spines, and has for its lateral boundaries the left lower scapular angle, and a vertical line situated about two inches to the right of the spinous processes. At its left extremity it runs to a point, but its right border is rounded. I will not trouble you on the present occasion with a detailed account of this interesting dulness, but simply point out once more that the "lower dorsal dull patch," which I regard as a test for the presence of an excess of pericardial fluid, is situated entirely below the level of this area.

The splenic dulness which you generally study in the flank is also to be traced in the back. It is usually the least obvious of the dulnesses we are now considering, and therefore the more likely to be overlooked and to give trouble in any exceptional case. The extent of surface which it occupies at the outer base varies with the variable size of the

organ, and its note varies to some extent with the degree of inflation of the abdominal viscera. I have not, however, noticed a conversion of this posterior dullness into a tympanitic resonance such as I have described as occurring in the flank.

The hepatic dullness needs a more complete description than time will admit to-day. You should remember as its leading features first its general shape with a broad end to the right and a pointed end to the left, and in the second place its level, which is immediately below that of the cardiac dullness, and which corresponds to the level of the eleventh and twelfth dorsal spines.

Urine Diagnosis of Typhoid Fever.—A. Robin ('Bulletin médical,' October 13th, 1897; 'Revue des sciences médicales,' July 15th, 1898) considers that certain characters of the urine, without being pathognomonic, may yet be utilised as a means of early diagnosis of typhoid fever. Their absence, however, does not negative this diagnosis. These characters are: 1. A colour of beef bouillon with greenish reflections. 2. Albumen in moderate quantity. 3. Disappearance of the urohæmatin. 4. Presence of indican. 5. Persistence or augmentation of uric acid. 6. Absence of uroerythrin. 7. Notable diminution of the earthy phosphates. Alone, each of these changes is valueless; but when they are observed together they are more or less conclusive.—*N. Y. Medical Journal*.

Forcible Correction of Spinal Deformities, by Stages, under an Anæsthetic.—Dr. V. P. Gibney, of New York, reported in this paper five cases which he had subjected to this method of treatment. He had been particularly astonished at the almost total absence of reaction after operation, the chief difficulty in the after-treatment being the keeping of the children in bed. Many of the patients could be hyperextended without the use of an anæsthetic, and in time an almost complete recession of the deformity could be secured in this way. The old idea, that one must endeavour to secure ankylosis as speedily as possible, seemed in the light of these recent experiences to need considerable revision, and certainly the latter offered a most cogent argument in favour of early and exact diagnosis.—*Medical Record*.

CHAPTERS FROM THE TEACHING OF DR. G. V. POORE.

No. IV.

GENTLEMEN,—I have to continue to-day with some of the laws which concern you as members of the profession, and I may perhaps call your attention to the fact that in 'Churchill's Medical Directory' you will always find a very good summary of these laws. I only bring to your notice such laws as affect you as general practitioners. If you take service under the Public Health Laws or Lunacy Laws you will have to become acquainted with very voluminous Acts of Parliament. I shall not trouble you with those here. I shall only deal with those Acts which concern us all.

The practice of anatomy is controlled by an Act of Parliament. I alluded yesterday to the fact that the early surgeons and physicians had the right to acquire the bodies of executed criminals for anatomical purposes. Partly from this cause, *i. e.* that dissection was made, as it were, to add to the disgrace of capital punishment, and partly from a too literal interpretation of the doctrine of the Resurrection, dissection of the human body was for a long time looked at askance by the public. The difficulties of obtaining bodies for dissection were at one time very great, and these difficulties led to the practice of "body-snatching." Body-snatchers rifled newly-formed graves, and took the deceased to the anatomists, who bought the body and asked no questions. The practice of body-snatching was supplemented, unfortunately, by murder, both in London and in Edinburgh. By the investigation of these crimes the public were at length aroused to the fact that an Anatomy Act was necessary. I will not give you all the clauses of the Anatomy Act, because you will not want them, not being professors of anatomy, but will merely state that facilities are given by it for the use of unclaimed bodies in public institutions. Sometimes people have left their bodies to be dissected. Now I would like to say with regard to these, that when people make wills of that kind it makes no difference to them, but it matters a great deal to the friends; and to make a will that the disposal of your dead

body is to be by some unusual method, is not kind to your executors. But the executors are not bound to carry out such directions as to the disposal of the dead body. It has been laid down in law again and again that there is no property in a corpse, and if in carrying out the directions of a deceased person as to the disposal of his dead body you incur any expense, the executors are not bound to pay it. There is a very interesting case bearing on that point which I will read to you.

Williams v. Williams. Chancery Division, March 8th, 1882, before Mr. Justice Kay. Miss Williams sought to recover £321 from the executors of one Crookenden, being the expenses of removing Crookenden's corpse to Italy for cremation. Crookenden made a codicil to his will dated April 12th, 1875, directing that three days after his death his body should be delivered to his friend Eliza Williams, to be dealt with by her in such manner as he had directed in a private letter, and further directed that a certain Wedgwood jar should be given to the plaintiff to be used as he had directed in the private letter; and he further directed that any costs to which Miss Williams might be put should be paid out of his estate within three months. The letter was as follows, and was dated March 28th, 1875:—"Dear Eliza,—I have written to be executed at some future time, a codicil containing my little request to you as you wished, the prints and the pots, and two little money legacies to servants. I have directed my body to be given up to you as soon as conveniently may be after my death. You know my wishes, but I here repeat them that you may have this letter to produce if need be. I should like to be burnt when I am dead on a, or rather under a pile of wood. I think the most convenient way would be to lay the body on a layer of dry faggots, and to build upon and around them a considerable mass of firewood, what is called cord-wood in some places, then to set fire to the faggots. If the fuel is only tolerably dry, it will burn with great fury for hours, and consume whatever is combustible. The calcined bones and fragments of bones can easily be collected when the ashes are cold, and I should like them put in the Wedgwood vase I have named in the codicil; if this would not hold them, then in any other larger vase which my

collection may contain, or which could be procured on purpose. Any would do, provided it were not metallic, or if metallic, not precious. You know my preference for earthenware. With reference to the ultimate disposal of this vessel and its contents, I leave that entirely to you. You might like the vase to be buried with you when the time comes, or you might prefer giving it a totally different destination. I should like to have written you a letter on a more cheerful subject, but as I want this done when it comes to the time, I will say no more than that I am your truly obedient friend and faithful servant, Henry Crookenden. You had better put this by and keep it." There was a nice little errand to ask a spinster to execute! It appears that Miss Williams did not build the funeral pile herself, as she was directed to, but she got the body exhumed, and got permission for that exhumation under false pretences from the Home Secretary. She then exported that body to Italy, where there was a crematorium. The body was cremated, and she was put to £321 expense. She brought an action against the executors for recovery of the amount, and it was disallowed by the Court. That is one of the most striking cases. The directions were very explicit, and there are many people who think that the directions in a person's will are in some sense sacred. I should say it is a cowardly thing to make a will of that kind.

Now just a word or two as to the relation of the doctor to the patient. A doctor and his patient are brought together under circumstances which involve great intimacy; and the doctor holds a position of trust and confidence in reference to his patient, and the law is very jealous that that confidence shall not be abused. A lady wished to benefit her doctor, and she proposed to him that she should leave him a certain property. However, he persuaded her that it would be better to give it to him by a *deed of gift*. She did so, and after the lady's death the executors disputed the deed of gift and the court disallowed the deed. They held that there had been an abuse of confidence, and that no doctor had a right to advise a patient for his own interest and against the interest of her relatives and friends. The court further stated that if there had been a will she might have altered that will thereafter by a codicil or otherwise, but a deed of gift was irrevocable.

Now a doctor is liable to actions for assault, and you must remember you cannot do anything to a patient without that patient's permission. If the patient be under age, then the permission of the parent is enough. A case which bears upon this point is that of *Latter v. Braddell and Wife and Another*. This case was tried in the Court of Appeal on February 24th, 1881, before Lords Justices Bramwell, Baggallay, and Brett. It appears that Latter was a servant girl in the employ of the Braddells, and that in October, 1879, Mrs. Braddell, suspecting Latter to be pregnant, accused her of it, and, on her denying it, had her subjected to a medical examination by Mr. S. The girl brought an action for assault, which was tried at the assizes before Mr. Justice Denman, but as the jury were unable to agree upon a verdict they were discharged. The case was next tried before Mr. Justice Lindley, who withdrew the case as against the master and mistress from the jury, and as against the surgeon he instructed them that they must be satisfied "that the girl had been overpowered by force, or by threat or terror of actual force." The evidence, however, went to show that the girl never objected to take off her clothes, and that she lay down and was quietly examined. Mr. Murphy urged, on behalf of the plaintiff, that the fact of the girl neither struggling nor forcibly resisting was no more proof of consent than a schoolboy holding out his hand to be struck, but Lord Justice Bramwell upheld the instructions given to the jury by Mr. Justice Lindley, and thought that there had been no evidence that the girl's wish had been overborne by violence or threat. The doctor, his lordship added, had appeared to have acted kindly throughout, but he dropped a word of warning to the effect that in such cases the wish of the master and mistress was no authority in the eye of the law for a doctor to examine a patient against her consent. It appeared to his lordship to have been rather a high-handed proceeding throughout.

Then with regard to restraining patients. The other day I found a patient who had just been admitted into my wards in a state of very considerable excitement, she wanted to go out of the hospital. She was not fit to go out. I told her so and that she would die if she persisted, but I took care to tell her that she could go if she wished, but that it was contrary to my advice, and that

such a course would be fraught with great danger to herself. The sister of the ward also spoke to and reasoned with her in the same way, and we succeeded in persuading her to stay, and she is under my care still. You cannot say "I forbid you to go," or to do anything which could assume the form of a threat.

There is now an Act of Parliament bearing upon the notification of infectious diseases, and that is an Act with which you all ought to be familiar. This Act, which was passed in 1889, makes it obligatory to notify to the Public Health authorities certain infectious diseases. The infectious disease must be notified by the head of the family, and further, "every medical practitioner attending on or *called* to visit a patient shall forthwith, on becoming aware that the patient is suffering from an infectious disease to which this Act applies, send to the Medical Officer of Health for the district a certificate stating the name of the patient, the situation of the building, and the infectious disease from which, in the opinion of the medical practitioner, the patient is suffering." I call your attention to the words "the medical practitioner attending on or *called to visit* shall forthwith notify." It happens to some of us to be called in consultation to these cases, and we have to remember that we are bound to notify. I may say I am always careful to inquire whether the case has been notified. If I am answered in the affirmative I usually do not take any further steps about it. But remember that the law says you are bound to notify. Sometimes this is of importance. For instance, supposing a doctor in attendance believes the case to be not notifiable, and the consultant comes to the conclusion that it is notifiable, and there is a difference of opinion, it would then become the consultant's duty to notify it, otherwise he will get into trouble.

The Vaccination Laws are now in process of amendment, and as we do not quite know what they are at present, I shall say nothing about them. Inoculation with smallpox is a misdemeanour which is punishable, and I need hardly remind you that the procuring of abortion is a felony, and if death results from the attempts to procure abortion, the crime is murder.

Finally one may allude to malapraxis. What is malapraxis? Malapraxis is the wrong or bad treatment of a patient, and every doctor is liable

to have an action brought against him for malapraxis, or even to be indicted as a criminal for having killed a patient by malapraxis. The law does not expect a doctor to show the highest possible skill; it expects him, however, to be able to prove to the jury that he showed a reasonable amount of skill and knowledge, and gave a reasonable amount of attention. A doctor promised to attend a lady in her confinement, and he promised to visit her on a certain day. Instead of so doing he went hunting. Labour came on, and the child was born dead. In that case the court decided that there had been a neglect of duty. A very good instance of a civil action for malapraxis occurred some years ago in connection with one of our big schools. A father took his son to school and put him in the house of one of the masters. There was a verbal agreement that under no consideration should the boy sleep elsewhere than in the house of this master. Scarlet fever broke out in the master's house, and the lad in question was one of the first to suffer. He was moved to the infirmary, where he died, and it was alleged that the infirmary, when he was moved to it, had not been properly warmed, and was not a fit place for the reception of a scarlet fever patient. The father brought an action against the head of the school and the doctor. The head of the school was accused of breach of contract, because, having promised not to move the boy out of his house, he did move him into the infirmary, and the doctor was accused of malapraxis because he had moved a scarlet fever patient to a place which was unfit for his reception. In this case, of course, there was a great deal of evidence on either side. The derelictions of duty, however, were very slight. The head master could not keep a case of scarlet fever amongst a number of other boys, and the boy was obliged to go to the infirmary, and whether that infirmary was cold or damp or not was not very clear. The verdict of the jury was for the defendants.

Another good instance of malapraxis which I should like to bring before you is an important one, and very well known. It is that of the *Public Prosecutor v. B. and K.*, who were medical practitioners. In the autumn of 1882 Mr. K. was called to attend a child of Mr. and Mrs. W., suffering from laryngeal obstruction. After con-

sultation with his partner, Dr. B., tracheotomy was decided upon, and the operation was successfully performed without chloroform, and with the result of giving temporary relief. After the operation a diphtheritic patch appeared on the pharynx, and the case ultimately terminated fatally. Shortly after the operation the tube became obstructed, and the father was asked to remove the obstruction by suction with the mouth, which he did. He contracted diphtheria (in consequence?), and he brought a civil action against B. and K. to compensate for his suffering and loss of time. The jury could not agree, and were discharged without giving a verdict. Next a criminal indictment was laid, and this was taken up by the Public Prosecutor. The charge was that of manslaughter through gross and criminal negligence (1) in not taking the temperature with the thermometer, (2) in not giving chloroform, and (3) neglecting to order proper diet and stimulants. These charges were preferred at Bow Street in November, 1883, but the magistrate (after five days' hearing) dismissed the case, reflecting that it savoured more of persecution than prosecution.

Why the matter should have been taken up by the Public Prosecutor is difficult to understand. But the case did not end there. We next find *W. v. B. and K.* in the Queen's Bench on May 8th, 1884. W.'s child had diphtheria in September, 1882. Tracheotomy was performed by defendants. The plaintiff was asked by defendants to remove some false membrane from the tube by suction, which he did, and afterwards suffered from diphtheria. The plaintiff sought to recover damages for suffering and loss of time, and alleged that defendants ought to have warned him of the danger he incurred by sucking the tube (there you come to something which perhaps has reason in it), and further alleged that, at the time of the operation, the defendants were ignorant of the nature of the case and that they had mistaken it for "ordinary" croup and not "membranous" croup, which is the same thing as diphtheria. Lord Coleridge in the course of the trial said that "a mere mistake would not be sufficient cause for action." A medical witness for plaintiff said that "he had once sucked a tube but would not do so again."

Lord Coleridge.—Unless it was in a case of any one very near and dear to you?

Witness.—Of course if it were my own child I should not hesitate to do it; that is a question not of science but of natural affection.

Lord Coleridge.—And I think so too.

The jury stopped the case and gave a verdict for defendants.

Now with regard to criminal malapraxis. One cannot lay down absolute rules and principles in these matters, and the best thing to do is to give you a few illustrative cases. The next one I have is a very important one.

Regina v. P. On March 3rd, 1875, at the Warwick Assizes, before Lord Coleridge, Mr. P. of N. was sentenced to six months imprisonment for the manslaughter of Mrs. Ann W. It appears that the deceased died during her confinement with her eleventh child. For her first nine confinements she had been attended by midwives, The prisoner attended her in the tenth and eleventh. On the tenth occasion forceps were used, and again on the eleventh. Evidence went to show that after the child was born he remained "doing something" which gave great pain to Mrs. W. He was seen to remove something, which he cut off with scissors and took away to the privy. He ordered brandy for his patient, and left saying she could not possibly live. The suspicions of the husband were aroused, and the substance in the privy proved to be fifteen feet of intestine, chiefly ileum, with portions of mesentery and parts of the cæcum and colon. At the post-mortem the vagina was found ruptured and the intestines were minus the parts found in the privy. The coroner's jury had found a verdict to the effect that the deceased died of ruptured uterus (which was not the case), and that Mr. P. was free from blame. As this verdict did not satisfy the neighbours the case came before the magistrate, and so to the assizes. It was probable that the vagina was ruptured by the forceps, and that the intestines had been mistaken for the cord, and had been pulled upon. It is not probable that so much intestine could escape spontaneously without rupture of the mesentery. There was much conflicting evidence.

This was a gross case, and one cannot say that the sentence of six months imprisonment was unjust.

There is a case of malapraxis against a nurse which is interesting. There was a girl in one of our big hospitals with pulmonary tuberculosis.

She was of a neurotic temperament, and the nurses took the idea into their heads, or some of them did, that she was what was called "hysterical." One morning this girl passed a motion under her into the bed, and the nurse in attendance apparently scolded her for it. It was necessary to give her a cleansing bath. She took her out of bed, and took her rather forcibly along the ward, and put her in a bath in the adjoining bath-room at an early hour in the morning, when the water, which was usually hot, was not hot. It appears that this patient sat three-quarters of an hour in water which certainly was not hot, but was more or less cold. As she came back to her bed she was noticed to limp, and the evidence went to show that the nurse treated her rather unkindly, telling her to walk properly, and that she could do so if she liked, and so forth. Before many hours had elapsed she died. Post mortem a mass of crude tubercle was found on one side of the brain; the incontinence of the bowels was due to a cerebral lesion, and so also was the limping on one side. That nurse got three months imprisonment. That is a very important case. The doctors there were quite free from blame; the nurse acted upon her own impulse entirely. I will not say hysterical patients never dirty the bed, but you may almost take it as an axiom that if you find that an adult patient has passed a motion in the bed you will do very wrong to put that down to hysteria. In this case the passing of the motion in the bed was due to a coarse lesion in the brain, and if any physician had seen the case at the time there is no doubt that it would have been recognised.

Next I will mention the case of *D. v. A.* that occurred in 1885. It came forward in the Court of Appeal, before Lords Justices Cotton, Lindley, and Fry. D. practised medicine at R., but was not legally qualified. M. became his assistant in November, 1883, and entered into a covenant not to practise in R., or ten miles round, without the written consent of D. The agreement between D. and M. terminated in August, 1884, and M. commenced practice in R., whereupon D. brought an action to restrain him. This was tried before Mr. Justice Pearson, who granted an interim injunction to restrain M. from practising. In the covenant between plaintiff and defendant D. was described as a "medical practitioner." Lord Justice Cotton said that "the covenant had been

broken, and the Court did not look with favour on the conduct of the defendant," but since the plaintiff had been practising as an apothecary contrary to the Act of 1815, George III, cap. 194, he could not enforce the covenant by injunction. Lords Justices Lindley and Fry concurred.

We are sometimes guided by American decisions, and there has been a decision in an American court that a contract by a medical man to attend a patient injured in a railway collision, and to be paid according to the amount of compensation recovered, was void. It is allied to the solicitors' actions I was talking to you about the other day.

Finally, never give an anæsthetic to a female patient without a witness being present. That is very important. Actions for criminal assaults have been brought against medical men, surgeons, and dentists, again and again by women who alleged that having been anæsthetised they were then violated, and that kind of accusation is not at all uncommon with an hysterical woman, and it is very difficult to rebut when only the man and woman are present. It is the man's word against the woman's word, and it is a very unsatisfactory state of affairs.

This finishes the legal part of our subject, and at my next lecture we shall begin the strictly scientific portion.

The Urethral Length as Diagnostic of Prostatic Enlargement.—Dr. E. L. Keyes ('American Journal of Medical Sciences,' August) sums up a paper read before the American Association of Genito-urinary Surgeons as follows:

1. The urinary distance varies in the adult healthy male from something over six to something under ten inches, but may be honestly averaged at eight inches.

2. The shorter lengths are found in short individuals having a small penis. A large organ naturally contains a long urethra, and this is most certainly the case if the individual be tall.

3. The age of the individual seems to cause a very moderate increase in the urethral length, irrespective of disease, or perhaps even of individual size.

4. In prostatic hypertrophy the urinary distance averages more than eight inches, and is longer in cases of peripheral general hypertrophy than where the enlargement is median, or in cases of bar.

5. In a doubtful case a consideration of the urinary distance may become an important element of diagnosis.—*N. Y. Medical Journal.*

DEMONSTRATION OF CASES

AT THE

NORTH-WEST LONDON CLINICAL SOCIETY,

Dr. MILSOM in the Chair.

MR. TURNER showed a girl who had been the subject of dental cysts. The case, which he would have shown before operation if he had been able, illustrated the course of dental cysts. A cyst had been present in the lower jaw on the right side and in the upper on the left side, and dental cysts were frequently multiple. Two years ago she had a swollen face, and an abscess connected with the first right lower molar, which subsided. Following that a painless swelling appeared, which eventually became so large that she sought assistance. When he saw the case there was in the alveolo-buccal sulcus a permanent swelling fluctuating all over, but with an edge of bone to be felt at the periphery. There was no sign of inflammation, and the alveolus was healthy except that the teeth were decayed and the first molar was dead, that is to say the pulp had died and putrefied. The inner plate was bulged below the level of the alveolus. There was a limited area of anæsthesia extending along the left alveolo-buccal sulcus and over the skin area of the inferior dental nerve. When operating he found a large cavity in the horizontal ramus filled with clear tenacious fluid, with crystals of cholesterin in suspension. No tooth fang projected into the cavity, which extended from beneath the second molar behind to beneath the first bicuspid in front, and the teeth which were undermined had lost sensation to heat and cold, but those in front were still sensitive. On the left maxilla, in connection with the first molar, there was a much smaller swelling, which appeared to be entirely bony. There was no history of pain or toothache on that side, but the tooth was dead. He found a small cyst which had already opened up the antrum. Microscopical examination of the cyst walls showed epithelium in both cases. It was important to note that dental cysts always occurred in connection with dead teeth, and hitherto had been found only in connection with teeth of the permanent series

Mr. Turner proceeded to minutely describe the course of typical dental cysts. As regards diagnosis they were often difficult to recognise in the early stage, that is before the bone was so thinned that there was crackling. Sometimes these cysts in the early stage were so hard that he had seen them mistaken for exostosis. In the same way they might be mistaken for fibromata. He advised exploration before proceeding to the operation, because the case was doubtful even with a dead tooth present or a history of dead teeth having been extracted. In the upper jaw after the age of fifteen or eighteen any swelling which was really bony or wanting in clinical signs of inflammation could not be a chronic abscess. For some reason the periosteum of the upper jaw was not inclined to deposit fresh bone on irritation beyond the age of about eighteen. At a later stage, in either jaw, a swelling which was painless, of progressive growth, over which the mucous membrane was moveable, and which to the feel presented either (1) distinct giving, (2) crackling, (3) fluctuation at the most prominent part, crackling around, (4) fluctuation all over with a bony edge at the periphery, could only be cystic; and a dental cyst was the commonest kind. Cystic disease of the antrum he had never seen producing clinical signs, and a dentigerous cyst was a disease of young life; while dental cysts occurred at any age, and were connected most often with an absent tooth, though possibly with a supernumerary tooth, on the other hand, a dental cyst might be met with, the originating tooth of which had been extracted (extraction of the tooth generally had no influence on the course of the cyst), while a retained tooth gave rise to a suspicion of dentigerous cyst. He had seen such a case. It was to be remembered that a dental cyst might inflame and suppurate, and was then, of course, painful, but the presence of actual tumour growth excluded mere abscess. For treatment he urged free excision of the outer wall and scraping away of the lining membrane, both as shortening the time taken in levelling up and as freeing the patient from an epithelial growth, for all these cysts were lined with epithelium.

Dr. ALEXANDER MORISON showed a girl the subject of double mitral disease. She had rheumatic fever when she was ten years of age. She had a presystolic bruit audible in the usual pre-

systolic area, and a double murmur at the inner side of the apex. An exocardial frictional murmur was also present at the left base. He had asked the patient to come in order that he might point out a condition which he thought had not had its due attention, namely, the usual position in which pulmonary congestion in connection with cardiac failure first manifested itself. In bronchial catarrh and crepitation associated with cardiac failure, evidences almost invariably were present first in the left lung. That was presented by this patient in a remarkable degree; only a fortnight ago, when the patient came to the Marylebone General Dispensary, the whole of the left lung, both anteriorly and posteriorly, was freely crepitating. She was sent home and put to bed, and in a week, the house surgeon having looked after her, the condition subsided. Anatomically the conditions were different on the two sides. There was a larger lung on the right side, and a longer course for the vessels from the right lung to the left heart than upon the left side. There was, however, a greater fall in the blood towards the heart from the right lung. On the left side the vessels were more dependent than on the right. In short, it was his opinion that the hydrostatic conditions accounted for the precedence taken by the left lung in the manifestation of evidences of stasis in connection with cardiac disease. The patient had done fairly well under digitalis and strophanthus, and of course she led as easy a life as possible. One usually found that the major congestion was *finally* at the right and not at the left base. He would like to hear if any members present had noticed the same thing.

Dr. Morison next showed a man *æt.* 59, the subject of tachycardia. He had never had any serious illness, and had been a hard-working man all his life. When he first presented himself at the Great Northern Central Hospital he was suffering from *œdema* of the legs, swelling of the face, enlargement of the cardiac area, and the usual evidences of dilatation, but without any distinct valvular incompetency. He had no kidney disease. He had since lived a comparatively easy life, and chiefly on that account had been able to pull himself together somewhat. The reason he asked the patient to come this evening was that the cardiac irregularity had practically resisted treatment. Two months ago he was taken into the

Great Northern Hospital, where he had rested for about a month, and had had hot salt baths, and regularly took every night small doses of calomel for the same period. During that time he expressed himself as better than at any time since the condition commenced. The heart-beats had ranged up to as many as 168 per minute. He had had a sudden drop in his urinary function, and since that had appeared he had been taken off the stimulating treatment with digitalis &c., and had been taking citrate of potash. No doubt the central fact in the vast majority of cases of irregular heart's action was the condition of dilated heart. A dilated heart implied an excess of blood in the heart cavity, an excess of stimulation of the surface of the heart internally, and that naturally brought into play the two great and constant factors of retardation and acceleration. Constant dilatation tended to constant retardation, and that constant retardation provoked constant acceleration. The object, therefore, should be to diminish the cardiac cavity and diminish the pressure within the ventricles. In a certain proportion of cases no doubt there must be some chronic disorganisation of the cardiac plexuses at the base of the heart, but these plexuses have very considerable vitality and are very hard to subjugate, and the innervation *on one side* seems to be quite capable of running both sides. It was interesting to see the improvement which had occurred to this patient under the hot salt bath treatment. A great deal of nonsense had been talked about the Nauheim treatment; the enormous hearts and enormous shrinkages described were, he thought, non-existent. But salt baths seemed to have a good effect, especially upon cases which were not associated with arterial sclerosis, and upon some which were.

Dr. HARRY CAMPBELL said in reference to Dr. Morison's second case that the presence of arterio-capillary sclerosis bore out the observation of Sir William Broadbent that in all cases of dilated heart, without valvular disease, there was high tension in the first instance. That was why calomel was so useful in such cases. In his experience irregularity of the pulse had very little influence on the prognosis; the great thing to go by was the pulse-rate. Regarding the alleged occurrence of passive congestion in the left lung before the right, Dr. Campbell did not think this could be explained

by the direction of the blood-vessels in the root of the right lung; he did not think this would influence hydrostatic pressure, since fluid pressure is conducted equally in all directions. He would have expected the congestion to occur in the right lung first, owing to the tendency of sufferers from heart disease to lie on the right side rather than the left.

Dr. CLAUDE TAYLOR asked whether Dr. Morison regarded tachycardia as having any bearing upon the prognosis, especially as to the mode of termination of life, *i. e.* whether apoplexy, or cardiac failure, or any other means of terminating life was more likely when the pulse was very rapid.

Dr. MORISON in reply said he was inclined to look upon tachycardia as, in a measure, a gauge of dilatation for the reasons he had given in his previous remarks. He thought too little importance was ascribed to the occasional irregularity of the heart which was found in fagged and worn-out people. A man came with no great heart-rate but complained of occasional intermissions or "stops," as he called them, and one was inclined to say it was his stomach or liver or something of that kind. He believed that in nine out of ten of these cases the patient had abnormal cardiac dilatability if not actual dilatation, however active distinct irritants in his intestinal tract might have been in provoking it. He did not think these cases were merely, in the absence of a rapid heart, to be dismissed as of little account. As regarded the evil influences of abnormal heart-rate, there were two extremes. The excessive bradycardia was the most fatal form of heart disease there was. As regards the consequences of tachycardia, he had been consulted by a woman aged fifty some time ago, with hard arteries and prominent eyes, a quick heart, and large thyroid. She had had a great deal of trouble, and he incidentally remarked to her friend when the patient was out of the room, and who was very inquisitive as to prognosis, that the condition might possibly lead to apoplexy. He learnt two or three months afterwards that the lady *had* died of apoplexy.

Mr. MAYO COLLIER showed a man aged 70 who had come to him three weeks before with a fractured patella, with a separation for five inches and the joint full of blood. He showed the case to exemplify the method of treatment in these latter days. He cut down on to the joint and laid

the two portions of bone bare, pressed out the blood from the joint, drilled a hole through the lower portion of the patella and passed a silver wire in, and with a curved needle passed it through the upper portion of bone, and then drew it together. The whole patella was joined, and there was now perfect union. He had only been kept in bed a fortnight. In some hospitals patients treated in this way were turned out the same day. They could see that the man could hold the leg out straight. A younger man could have been discharged in two or three days.

Mr. COLLIER showed next four young women who had been the subjects of chronic deafness of long standing. One had been deaf seven years with a chronic discharge from both ears and caries of the petrous bone. The most important part of the treatment consisted in removing the chronic obstruction of the nose, which allowed the congestion of the Eustachian tube to subside. Consequently she had now perfect hearing, as tested by her readily answering questions addressed to her in a normal voice. The next case was one of considerable rhinitis with obstruction and pharyngitis. He operated upon those conditions, and the hearing was now very much improved. He held that these cases were entirely due to nasal obstruction, which kept up chronic congestion of the Eustachian tube, consequently air was not allowed to enter the tympanic cavity so as to maintain the equilibration of pressure, and various troubles ensued from that. In the third case the ossicles were ankylosed and there were adhesions, so that the result was not quite so perfect as in simpler cases. The next case had chronic rhinitis and nasal catarrh and obstruction. She had deafness of a very marked character. He had not operated upon her, but simply treated her with a lotion, which reduced her nasal catarrh, and her hearing was immensely improved. He had recently sent a paper to the 'Lancet' wherein he was anxious to show that the very essence of treatment of ear disease was the treatment of the always present nasal affection. If there was Eustachian obstruction that was followed by deafness due to un-equilibrated pressure in the tympanical cavity, then followed catarrh, then pus into the cavity, then discharge in the ear. He brought these cases forward as selections from his clinic of the previous day.

Mr. Collier next showed a man the subject of macroglossia. The tongue was so large that he could not speak, and his life was therefore miserable. Someone had extracted the whole of his teeth to give room for the tongue. It was a gradual swelling of the tongue. On the right side was a nasty-looking slough, and the whole was very suggestive of gumma of the tongue. He looked upon the condition as undoubtedly due to syphilis. He suggested local mercurial fumigations. It was a very marked case of syphilitic glossitis.

Mr. Collier also showed a young woman the subject of laryngeal obstruction. The case, he said, illustrated how the ravages of syphilis might be unnoticed by the patient or by her medical attendant. She came to him with the whole of the upper part of the larynx gone, and the space between the epiglottis, arytaenoid cartilages, and epiglottidean folds contracted to the diameter of a lead pencil. The whole of the epiglottis had gone. Behind the epiglottis she had a hole which a pencil could go through. She had never noticed anything the matter with her throat. On first seeing her she had caught cold and the obstruction to breathing was so severe as to be dangerous, and tracheotomy had to be performed to save her life. The tracheotomy tube she had now worn for five years. He had tried to open the obstruction in the upper part of the larynx, but the cicatricial tissue was of such a character that it behaved like a potato after it was cut, namely, the slit made by the knife remained sharply defined, and simply healed straight away, showing no resiliency. He had therefore to be content with the subsidiary opening in the trachea, and she seemed to be going on very well. They could see that she now breathed quite easily and without any distress.

Dr. CLAUDE TAYLOR related a similar case which occurred to him while he was house surgeon to Mr. Symonds in 1895. It was evidently a syphilitic stenosis. It was useless to try any operative measures in the larynx, and all that could be done was to put in a tracheotomy tube and leave it there for the rest of the patient's life.

Mr. COLLIER concluded the meeting by showing a woman of middle age who had been under his care four or five years. It was a recurrent case of polypi of the nose. Directly she got clear in the nose and free from the obstruction she absented herself from the hospital. He had long held that

removal of the polypi was not the only thing for such a case, and that the after-treatment was of much importance. The essence of polypoid degeneration of the mucous membrane of the nose was a previous hypertrophic rhinitis, and polypoid growths occurred as the result of congestion and irritation, just as warts would appear on the prepuce from the same cause. Her nose was now again completely obstructed on both sides, and there were dozens of polypi present. If she could only be induced to remain under treatment after the polypi had been removed, there would be no recurrence.

NOTES, &c.

Forcible Correction of Deformities in Pott's Disease.—Dr. F. E. Peckam said that this new treatment was naturally of the greatest service in the early stages of deformity, and that it was the treatment *par excellence* for paraplegia. In one case, in which he had attempted too much at the first operation, there was a severe and dangerous degree of shock for two or three days afterwards.

Dr. Henry Ling Taylor, of New York, said that while the extravagant claims made for this operation had not been realised, on the other hand the fears of some regarding the great danger of the treatment had been proved to be groundless. The result would probably be the adoption of a method of gradual reduction, together with better support for the diseased spine.

Dr. Newton M. Shaffer, of New York, thought that this forcible treatment was chiefly of interest in connection with disease of the mid-dorsal region, for in the lumbar region the disease could ordinarily be treated successfully without surgical interference. A good deal of the apparent increase in height was the result of a modification of the compensatory curves, and not of a decided change in the bone itself.

Dr. R. H. Sayre thought another danger associated with the forcible correction of these deformities was to be found in the possible rupture of an abscess into the lung or of the aorta. He seriously doubted the wisdom of using much force in the effort to reduce the deformity.

Dr. C. C. Foster, of Cambridge, said that the

spine could not be expected to throw out callus until the subsidence of the active tuberculous process, yet he thought a straight bone would unite just as well as a crooked one.

Dr. Judson was of the opinion that the revival of this old treatment would prove salutary by inducing surgeons to apply mechanical force properly and continuously.

Dr. A. M. Phelps, of New York, announced his intention of trying the treatment in fifty cases, and then awaiting the ultimate results. He thought that relapse would take place in all but the few cases in which the treatment was undertaken very early, and before a large kyphos had formed. The gap in the vertebral column would not heal by the deposit of bone, because fibrous tissue would grow over the ends of the bone. For this reason he was now wiring the spinal processes together, by drilling a hole through the upper vertebrae and weaving it in and out until the entire kyphos was firmly fastened together.

Medical Record.

Forcible Straightening of Spinal Curvatures under complete Anæsthesia, with a Report of Cases.—Dr. John Ridlon, of Chicago, presented in this paper his more recent experience with this method. He said that his first operation of this kind was done in June, 1897, and that since that time he had operated in sixteen cases of spondylitis, in seven cases of scoliosis, and in one case of rachitic curvature. Four cases had demonstrable abscesses, one case had an old sinus, and one paraplegia. There were no complications in these cases following the treatment—indeed, the paraplegia was positively benefited. In spite of the free use of felt padding and every precaution to prevent pressure sores, these had been of frequent occurrence. Although he had allowed only two of these patients to walk around, yet the deformity had returned to some extent in all of the cases on the removal of the jacket and traction. The risk involved in this new method of treatment seemed to him to be only that of general tuberculosis or tuberculous meningitis, just as there was after forcible straightening of tuberculous joints. This risk he estimated at 10 per cent. He thought the period of treatment would be actually lengthened by this method of forcible correction.—*Medical Record.*

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A DEMONSTRATION OF SURGICAL CASES GIVEN TO THE POST-GRADUATE CLASS, CHARING CROSS HOSPITAL,

May 12th, 1898.

By HERBERT F. WATERHOUSE,
M.D., C.M.Edin., F.R.C.S.Eng.,

Surgeon to Out-Patients and Lecturer on Anatomy,
Charing Cross Hospital.

CASE I.

GENTLEMEN,—The first case to which I desire to draw your attention is one of considerable rarity, and at the same time the result after the very severe injury is so good and the case serves as such a useful lesson from many points of view that I think I may very well discuss it at length with you to-day—compound comminuted fracture of the lower end of the humerus, with rupture, complete or nearly so, of the median nerve.

I shall divide the subject of my observations into two parts: (1) injuries to the elbow-joint; (2) injuries to nerves.

Towards the end of August last year this little girl was in a goat-chaise when it tumbled over and threw her out, the other occupant of the chaise falling on her. She sustained a very severe compound comminuted fracture of the lower end of the humerus. This skiagram which I show you was taken some days after the injury, and you will notice that it presents several points worthy of remark. In the first place the ulna and radius are clearly uninjured, and the photograph shows very well the epiphysis at the olecranon end of the ulna. If, however, you look at the lower end of the humerus you will see that it has been comminuted so extensively that the fragments can scarcely be recognised. Here we have obviously had a transverse fracture, but in addition we have had the lower fragment extensively comminuted—broken up into several bits, and, as you will see from the photograph, they are practically beyond

identification. I want you further to notice this very sharp edge of bone, which is the lower end of the upper fragment of the humerus; you can all see how it projects almost right under the skin. Its sharp, almost knife-like edge is a noticeable feature of the skiagram. Another point to which I wish to direct your attention is that this fracture was compound in two places. The father tells us that the ends of the bone were sticking out in two different places, and you will see the large scar above and a small scar below, but the lower one is comparatively unimportant. The upper one is of extreme importance. In addition to what I have related, there has been a very severe injury to the median nerve. The exact extent of the injury it is a little difficult to state. In all probability the nerve was almost entirely cut through by the sharp lower end of the upper fragment, because after the accident the child had undoubtedly complete median nerve paralysis. She is recovering from this, but the recovery is not by any means complete yet. I may demonstrate this point to you. In the first place you will notice, and indeed you can very readily feel under the scar a sharp edge of bone, which the skiagram enables us to say without doubt is the lower end of the upper fragment.

You would expect, after such injury, that there would be a considerable amount of stiffness and even that there would be almost absolute rigidity of the joint, and yet as a matter of fact, though the elbow-joint presents such an extraordinary appearance and though there has been such extreme comminution, the movements are almost perfect; extension is perfect, flexion is only slightly limited, and I take it it is really only limited owing to the presence of the great mass of bone thrown out to glue together the comminuted fragments of the lower extremity of the humerus, and if the excess of bone could be removed I have very little doubt that flexion would be practically perfect. I think you will agree that pronation and supination are also perfect. We further notice at the present moment that there is certainly some slight atrophy of the muscles of the forearm supplied by the median nerve. You will observe that the hand is very cold in the part supplied by the median nerve. On the tips of the index and middle fingers there are ulcers, said to have come without assignable cause, and are therefore what

are known as trophic sores. There is very marked atrophy of those muscles of the ball of the thumb that are supplied by the median nerve. The child cannot use her abductor pollicis, and there is partial loss of sensation over the digits supplied by the median nerve. If I touch lightly the skin with the point of this pencil, you observe that though she feels the contact in certain parts of the skin to which the digital branches of the median nerve are distributed, there are patches in which the touch is not perceived. She has, therefore, partial anæsthesia. The father, who has intelligently followed the case, tells us that the loss of sensation and the motor paralysis (and this is confirmed by the doctor who was attending her), which were at first complete, are both very markedly improving; that is to say, month by month, even week by week, a change for the better can be discerned. The skiagram shows us clearly what was the bone injury, and the result is to be considered as exceedingly satisfactory, even though the elbow has such an unsightly appearance. I am able to say so because I have not had anything to do with the treatment, the child having come under my observation some months after the receipt of the injury.

The question arises, what was the injury to the median nerve, and what treatment, if any, is now to be adopted? In the first place, with regard to the median nerve you will have no doubt that it was cut through or almost cut through by the sharp edge of bone. If it had been only bruised, recovery would have taken place long ago. You will remember, as the skiagram showed us, that we have a very sharp anterior margin to the upper fragment, which of course came up through the skin just in the position where the median nerve lies under the skin cicatrix, and I feel certain that the nerve must have been almost completely divided as by a knife. Probably, however, the division was incomplete, and the partially divided nerve ends were still held together by the nerve sheath, and some undivided strands of fibres. You will notice that as regards the flexor muscles of the forearm their function is nearly perfect. The only muscles we can find paralysed are the muscles of the ball of the thumb, and it is a fact that we usually find these are the last to recover, and frequently they only recover partially in cases of median nerve paralysis. It is the rule that the

finer and more specialised movements, and the more delicate appreciations of touch, are partially lost after division of the median or similar nerve; usually the power of the muscles which execute the coarser and less specialised movements is completely regained. I know of a case in which a man divided his median nerve by a fall through a glass skylight. I sutured it some three months after the injury, sensation has returned almost perfectly and the larger muscles have not wasted at all, but he tells me that he does not now enjoy anything like the facility he did in many movements, and though there is no loss of power he complains that he cannot now perform the fine movements so well as he could before.

Now let us look at the case from the point of view of fracture. The point to notice first is how comparatively slight an injury or fall has produced such an exceedingly severe comminution of the lower end of the bone. Of course we know that in order to have got anything like the result you see, the wound made by the accident must have healed perfectly aseptically, and no doubt it was treated antiseptically and skilfully. One point of very great difficulty in the diagnosis of fractures of the region of the elbow-joint is that swelling comes on so rapidly that within an hour or two it will obscure the diagnosis. In this case I do not say that the position of the bones is the best that could be obtained, but still the result is so excellent as regards movement that one does not like to find any fault with it, especially as I am sure that nobody could have guaranteed anything like so good a result if he had only seen the joint for the first time after it had become swollen and distended. Under such conditions it is, as I have said, difficult to say what has happened. What, then, should be done by way of treatment? One reason why such a good result is obtained here I am perfectly certain is that movement was commenced early; it was commenced on the seventh day. For some months past I have treated every fracture in the neighbourhood of the joint, more especially the ankle-, elbow-, and wrist-joints, by massage from the very first. In any fracture near the joint, where there is any likelihood of stiffness, I advise massage from the first day, first very gently once a day, and afterwards twice a day; and not only has union not been interfered with, but in spite of the movement I believe union has taken

place quicker than would have been the case had fixation been rigidly maintained. An important rule is "Do not mind so much about the accurate apposition of the bony fragments in any fracture very near a joint, but think rather of the subsequent mobility." The fact that stiffness does so frequently follow these injuries in the neighbourhood of joints is a very strong argument in favour of early massage, and I can assure you that early massage is really appreciated by the patient. By using massage to a fracture even on the day of injury, very little pain is caused.

The exact explanation of this is difficult to give, but of the fact there is not the slightest doubt. When I first heard of this treatment being carried out in Paris, I thought the patients would object strenuously, but as a matter of fact I never have met with a patient who complained that the pain was anything like unendurable. You will find even though at first massage is a little painful, that when the massage is once commenced patients are quite willing when their time comes round again to have the treatment carried out.

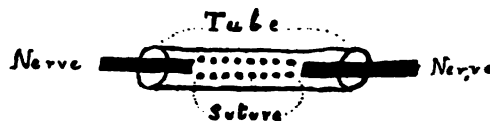
An interesting point is that the brachial artery has in this case escaped injury. I take it that the artery probably has, as arteries frequently do, slipped out of the way, and that the sharp margin which cut the median nerve has missed the brachial artery.

Now a few words with regard to the surgery of nerves and the treatment that should be adopted. We find that directly the nerve is completely divided across, the peripheral part degenerates; I think we may say that does so in all cases in its entirety. We find, as is still shown in this child, that the skin becomes colder, and there is marked anæsthesia, and in case of total division there is absolute paralysis of motion and sensation. We further find that the development of the new nerve-tissue is a most variable matter. I have seen sensation almost perfect a fortnight after I have sutured the divided nerve; on the other hand, I have known almost two years elapse between the time of suture and the time when it has reached the limit of its perfection. This child's accident happened nine months ago, and we find improvement is still going on month by month. Suturing nerves is frequently a most disheartening thing. I have often been discouraged in my earlier cases. I have cut down upon and sutured a divided nerve,

and sometimes three, four, five, and in one case six months elapsed before any real improvement occurred, except some slight return of sensation; no improvement whatever in motion being noticed. Then, perhaps, after six months movement slowly begins to return, and may eventually become almost perfect. I would therefore say that one must not give up all hope until two years have elapsed after the suturing was done. Another point is that in suturing nerves one must not expect in every case a perfect result. Recovery is usually only partial. Of course the most satisfactory results are those in which immediate suture is performed—that is to say, within a few hours of the division of the nerve. Secondary suture—that is to say, after the bulbous enlargement has formed on the lower end of the proximal portion—is not so favorable as a rule as primary suture; but after both primary and secondary suture the recovery is frequently only incomplete, the deficiency being chiefly in the matter of the finer and more specialised movements and sensations which I have just referred to. In the case of any incised wound the medical man should always test the sensation beyond the point of injury, to make certain that the nerve has not been divided. It is a precaution which is frequently omitted, as shown by the frequency with which secondary sutures are performed on nerves weeks or months after the receipt of an injury. There is nothing simpler than stitching a nerve. You have got to get your stitch into the nerve sheath, and then sew the nerve up just as you would sew an ordinary wound; there is no difficulty or complication. In the old days we were told not to pass the needle right through the nerve; now-a-days we invariably do it, and no harm results. But you should always suture a nerve with absorbable substance, preferably fine catgut; if you use horsehair or silk or silkworm gut you may get some amount of pain or irritation, as the non-absorbable suture may act as a foreign body.

In certain cases one finds that the ends of the nerve have considerably separated, and in one instance I found in a median nerve case the divided ends had retracted, and lay about one and a quarter inches apart, and that the upper portion was bulbous for three-quarters of an inch. When I had cut that off, as I was obliged to do, there was an interval of two inches between the ends. It is

not of much use in these cases to forcibly pull your stitches tightly; they will only cut out. I have found that much the best way to deal with such cases is to take a decalcified bone drainage tube, and to cut into it in its long axis, so that when the margins of this incision are opened a deep groove or trough is formed, inside which the nerve ends are placed, and when the margins are released the tube resumes its cylindrical form, and prevents any other tissues falling between the ends of the nerves, and in this way you are more likely to obtain union, the upper end growing down towards the lower. It is just as well in such



cases to put in one or two what the Germans call "guiding stitches," *i. e.* loose stitches between the ends of the nerve, so as to direct the new nerve-fibrils in their journey from the proximal to the distal end of the nerve. It is obviously better to employ these loose guiding stitches than to put in sutures which will certainly tear out. The decalcified drainage-tube remains as long as we require its presence in the tissues, and in four or five weeks at the longest it is completely absorbed, by which time we hope the new nerve-fibrils will have passed from one end to the other. It is well to remember both in suturing nerves and tendons that making use of flexion of joints almost invariably helps the surgeon considerably. When you are operating with the arm extended you may have an interval of one inch, but if you flex the elbow and then forcibly flex the wrist you may find that the interval has almost disappeared. This fact is exceedingly useful in suturing tendons; if a tendon is sutured on the dorsum of the hand or foot you dorsiflex, and approximate in this way the end as much as you can.

Another point to bear in mind about nerve suture with reference to its results is that certain cases undoubtedly fail, but complete failure is very rare. I have seen cases in which motion has returned and sensation has not. I have had others in which sensation has been regained, but no improvement in the power of movement has been obtained. Either or both may be absent.

Now a word further regarding the treatment of injuries to the elbow-joint. Only nine days ago a case was sent to me from the North of England for an opinion as to what should be done. The patient was accompanied by several skiagrams. There had been fracture of the internal condyle of the humerus into the elbow-joint. The arm, however, had most unfortunately been put up in a straight position (that is, with the elbow extended). The practitioner wrote me that when he flexed the arm he could not get the broken fragment to remain in proper position, but that he found he got better apposition with the arm extended. The fracture has united, an undue amount of new bone has been thrown out, ankylosis of the joint has occurred, and the patient now goes about with a rigidly fixed, extended elbow. A stiff joint would not have mattered so much if the elbow had been flexed, as an upper extremity with ankylosis of the elbow in the flexed position is a very useful member. In this case no movement was attempted until seven weeks after the injury. Under gas I tried to break down the adhesions, but failed. In such a case I think the wiser plan would be to excise the elbow-joint so as to get movement, because the patient in his present condition has an arm that he says is almost useless to him, owing to the ankylosis of his elbow-joint in the position of complete extension.

CASE 2.

I wish now to show you this infant in order to illustrate a somewhat simple and easily applied method of treating nævi of the lip, vulva, &c., which are very disfiguring. The age of this child is 8 months. She came three months ago to my out-patient department at the Victoria Hospital for Children with an enormous nævus of the upper lip. I show you the case in the course of treatment. When I first saw the child the lip was four times the size it is now; it was very hideous, and purplish in colour. Excision was absolutely out of the question, as the nævus involved the whole of the upper lip. Electrolysis would have been an exceedingly tedious process. It is quite true that almost every subcutaneous or mixed nævus can be cured by electrolysis, but it is a very tedious method of treatment, and is not practicable in busy out-patient work. The treatment in these cases which I now generally adopt

is that known as galvano-puncture. I take an electric cautery needle of good size, and make one puncture in the centre of the everted lip on the mucous aspect, and from this centre the needle is worked about in all directions, so that only the one puncture on the mucous surface need be made. Care must be taken not to approach the needle too near to the skin. If you keep the cautery at a dull cherry-red heat you will have no hæmorrhage, however vascular the growth may be; at that heat the cautery is one of the best hæmostatics, but at a white heat the power of preventing hæmorrhage practically disappears. On no single occasion have I had any hæmorrhage whatever. I have had to operate three times for this huge nævus, and probably I shall have to use the needle once or twice more before the whole nævoid structure is destroyed. You will see that this result can be obtained without any scarring of the skin. It is a method which is applicable to almost all nævi, and I do not think it is sufficiently generally appreciated. You will see that on the under surface some little piece of mucous membrane has sloughed, but this is only a tiny portion. The improvement in appearance is most marked. The one kind of nævus for which this treatment is not applicable is the cutaneous nævus or port-wine stain, because of course there one single puncture will not do any real good. Pure cutaneous nævi are always very unsatisfactory to treat. The advantage of the plan I have mentioned is that you can use the same puncture for successive applications. It is well wherever practicable to cover the part over with an antiseptic dressing as soon as the needle has been withdrawn. This child has other large nævi in other parts of its body; this is not unusual, nævi being frequently multiple.

CASES 3 AND 4.

Next I desire to show you two cases of nasal syphilis, which is a very common complaint. This first patient is a man aged 30; he came to my out-patient department on Friday, and almost directly he entered the room—certainly as soon as he spoke to me—I recognised the odour of nasal syphilis. I entertained little doubt that he had some necrosis in the region of the nasal passages. He had syphilis seven years ago. He is married, and has had a healthy child. Inside his nose he

has a marked ulceration high up on the septum of the nose; it has been troubling him eighteen months, and there has been an offensive odour eight or nine months. An interesting point is the diagnosis between syphilitic ozæna and true ozæna, though really the name ozæna should not be applied to syphilitic affection. In real ozæna the patient cannot smell the unpleasant odour which arises in his nasal chambers; however shockingly offensive it may be, the patient will be entirely unconscious of it, even if he or she be an educated and intelligent person. I have had two dental surgeons consult me telling me that friends complained that they had a foul-smelling breath, but at first they were sure they had not, because they could not smell it themselves; gradually, however, patients dropped off, and they then began to think something must be wrong. In both these cases the patients carried about with them the typically offensive odour of ozæna. Another point is that there is a great difference in the smell of ordinary and syphilitic ozæna. In syphilitic ozæna, on the other hand, the patient, at least in the earlier stages, is aware of the odour; but as the necrosis goes on the patient frequently partially loses his sense of smell.

In this case we find there is considerable necrosis. Now I warn you very strongly against the practice of pulling away a bare piece of bone in the nose before it is quite loose. This does not apply so much to the lower part of the nose, such as the inferior meatus, the inferior turbinal bone, the middle meatus, and even the lower part of the middle turbinated bone; but in the upper regions of the nasal chambers the procedure is distinctly a hazardous one. On two occasions I have known death occur from septic meningitis, from medical men hurriedly examining the nose, finding there was some necrosis in the region of the upper parts of the nose, and pulling the dead bone away. In one case the fragment was shown to me, and at least part of the cribriform plate of the ethmoid was pulled away. In the upper part of the nose I generally do nothing but spray thrice daily with a mild antiseptic lotion, and wait for Nature to cast off the sequestrum; she will cast off the bone in time without any risk whatever. Another reason why you should not pull at these parts is that you can never tell with anything like exactitude what are the limits of the necrosis. This man is having

iodide of potassium twenty grains three times a day. In nasal syphilis only large doses of the iodide have any real beneficial result. He is having, in addition, a mild antiseptic douche to the nose. You see this patient now brings to show you a large piece of one of the turbinal bones which has come away since he last attended at the hospital.

The next patient, also a young man, is another example of tertiary syphilitic disease. He has been under treatment only two days. He came to my out-patient room on Tuesday last with an opening in the roof of the mouth through which a dirty wash-leather looking slough was protruding; in fact, I at first thought it had been stuffed with iodoform gauze. I passed a probe, and found he had a very considerable amount of necrosed bone in the roof of his mouth. This is a very frequent condition—gummatous periostitis with necrosis of the hard palate. These cases are almost always exactly in the middle line, and if they are treated vigorously and antisypilitically they almost always recover when the bones have been discharged, and comparatively seldom leave a permanent opening. The books say openings are frequently left, but if large doses of iodide are administered there is often no aperture left in the palate. You may say this is not a case of nasal syphilis; it is apparently in the roof of the mouth, and yet most of these cases of syphilis in the roof of the mouth begin in the floor of the nose. I have watched several, and have questioned the patients closely and found that to be so. This man's case is an illustration of the same fact. He tells us he had some matter from the nose two months ago, whereas the mouth trouble dates from a week to-day. On examination we find he has a distinct healing gumma in the floor of the nose, whereas in the roof of the mouth the gumma is comparatively recent. There is very considerable necrosis.

CASE 5.

The next case is really a very interesting one. This patient is suffering from what I call, for want of a better name, traumatic gumma. I do not show him as anything very characteristic to look at at the present time. Ten weeks ago he had on the outer tuberosity of the tibia a perfectly circular swelling. The skin had broken over it, and for several weeks he had dirty wash-leather

sloughs. At first we could not get them away, and on one or two occasions I have cut the dirty sloughs away. The reason of this persistency was that mixed up in the gumma was a large amount of this strong fascial band of the deep fascia attached to the external tuberosity of the tibia. In this case there is a history of distinct injury. He slipped down a ladder and knocked himself on the spot where you see the lesion. There can be no shadow of doubt that he had a gumma, and a point of interest is that he had been treated outside without improvement, the syphilitic nature of the ulcer not having been recognised; directly he was put upon iodide he began to improve, and the lesion is not now a quarter of the size it was. He had syphilis six or seven years ago.

The special point to bring out in this case is that traumatism does very frequently give rise to a gumma; it determines the seat of the gummatous process. I believe this fact is not mentioned in books. An instance I very well remember is the following:—A young gentleman who had been dining too well got mixed up in a street row, and was taken to the police station. On the way he became obstreperous, and assaulted his captor violently, and the policeman struck at him twice with his truncheon; the man put up his arm to save his head from the blows, and received the blows on the forearm at two different points on the subcutaneous surface of the ulna. He was sentenced to four weeks' imprisonment, and when he came out of jail he came to see me, when he had a distinct ulcerating gumma on one of the spots where the truncheon had struck him, and on the other spot was a swelling which had not yet burst, and disappeared entirely under large doses (30 grains) of iodide given twice daily. He had had syphilis five years before. The conclusion was absolutely irresistible that the gummata had formed at the site of injury. This man before you is an equally certain instance of the same thing, and I have for three or four years past been on the look-out for these cases. I remember also the case of a professional football player who was kicked on the inner side of the upper end of his tibia, where he developed a gumma.

Another instance was that of a man who was attending here for tertiary syphilis. He was in the third year of his syphilis when he was run over by a cab and struck on the forehead. Some three

or four weeks later a big lump formed there which burst, and when I saw him some weeks afterwards there was a distinct gummatous ulcer which had resisted ordinary (non-syphilitic) treatment for some time. Directly he was put on iodide the gummatous sore healed.

I would wish clearly to point out that whilst I do not say that by any means all or even a large proportion of cases of gummata are associated with traumatism, still in a very considerable number of instances a gumma forms at the site of injury shortly after a trauma. The patient is predisposed to gummatous formation, and the injury localises the gumma.

Disturbances in the Evacuation of the Urine in Children.—Kutner reports the case of a boy aged 8 years, who since his sixth month of life suffered frequently with retention of urine. After excluding a urethral calculus and a congenital stricture, the diagnosis was made of a chronic reflex spasm of the internal sphincter of the bladder. Another case was that of a girl aged 7 years, who since birth had unconsciously voided urine every two or three hours. Examination revealed a remarkably distended bladder. The case was also a chronic spasm of the sphincter of the bladder, and is thus explained:—As a result of the transcendancy of the sphincter over the muscular power of the bladder, the evacuation of urine was hindered until the limit of the receptivity of the urine on the part of the bladder was reached, when the muscular walls of the bladder caused the evacuation of a certain quantity of urine. As soon as the intra-vesicular pressure became diminished the sphincter obtained again the transcendancy, and prevented the egress of additional urine. The cases are interesting because, although both depended upon the same cause—a functional disturbance of the sphincter—a spasm, the result was different in each case. In both cases dilatation of the sphincters resulted in complete cures.

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ON SOME FURTHER POINTS RELATING TO APPENDICITIS.

A Clinical Lecture delivered at St. George's Hospital

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A MATTER for careful consideration in dealing with cases of appendicitis is the proper period for operation if such be indicated. Speaking generally, it is no doubt best to operate if possible during the quiescent stage, or at all events after the acute symptoms of an attack have passed.

So far as the deliberate removal of the appendix is concerned, it should, I think, certainly be advised, for the reasons given in the previous lecture, if a second attack of the symptoms occurs.

In many cases it is, I have no doubt, greatly for the benefit of patients if the appendix be removed after the first attack, for although there are, as I have said, cases which undoubtedly get apparently well after one attack if left without operation, some certainly prove fatal during the second attack, and some, indeed, during the primary attack in which suppuration followed by perforation into the general peritoneal cavity may occur—the so-called fulminating cases. In the ordinary relapsing appendicitis the only means we have of distinguishing between the different cases clinically is by abdominal section, which in competent hands should subject the patient to so little risk, that it is in my opinion desirable in the majority of instances after a primary attack, if the least sensation of any abnormal kind remains upon the subsidence of the objective symptoms.

Although we speak somewhat loosely of a patient being "well" in the intervals between attacks, *complete recovery is in fact quite the exception*. There may, it is true, be no tenderness or pain, but the great majority of patients are conscious between the attacks of something unnatural in the cæcal region, this unnatural feeling as often as not amounting to nothing more than a sense of slight restraint or discomfort upon the patients stretching themselves out to the fullest extent, as, for example, in standing in an exaggerated upright position, or bending slightly backwards. This symptom is, I

believe, a sure indication for operation, to be performed deliberately for the purpose of removing the appendix.

In cases, whether the attack is primary or recurrent, in which abscess forms, the best means of dealing with the appendix is an interesting question. Should the abscess be merely opened or drained, or should the appendix be removed at the same time? In a general way there is no possible doubt that drainage only of the abscess is indicated unless the abscess be very small, in which case I prefer to clear the abscess well out and remove the appendix. Under such circumstances the operation should not be dangerous, at all events in my experience all the cases so treated have recovered, and the healing period has been greatly shortened. In the larger acute abscesses I have always been content with free drainage, excepting in two cases in which the appendix was presenting so obviously at the base of the abscess that it would have been foolish to leave it, and it was in each case easily removed without the interference with any important adhesion. The main reason, of course, for being content with free drainage is the danger of infecting the general peritoneum by the separation of protective adhesions if the operation for removal of the appendix be attempted. This objection, if the operation is performed by an expert, is, I think, overrated. There is, however, another reason for not subjecting these patients, who are often extremely ill, to the risk of removing the appendix, viz. that if they survive the immediate effects of the disease the abscess almost invariably heals perfectly with drainage—although the appendix has been left untouched at the operation—healing sometimes very quickly upon the coming away of a fæcal concretion or perhaps the sloughing off of the whole appendix, which I have washed out of the cavity as a gangrenous mass within forty-eight hours of opening the abscess in four cases. It is at first sight singular that so few cases of fæcal fistula or sinus follow upon the opening of these abscesses and other operations upon the appendix and cæcum. Permanent sinus or fistula after these operations is very rare, the only case which I have seen in my own practice in which a fæcal fistula has remained open for a very long period being that of a youth in whom the opening of an abscess at the angle of the scapula was followed by the

escape of a faecal concretion which was found to have come from the appendix. The operation for dealing with such an extensive condition in order to provide proper drainage was very severe, and even four years later a small unhealed sinus remains; but there is every reason, judging from the progress of the case, to believe that finally healing will occur. The inherent tendency of these wounds to heal not only shows that the removal of the appendix in these cases of acute abscess is generally unnecessary, but what is equally important, or more so, it very distinctly negatives the justifiability of the performance of extensive operations for the purpose of closing these fistulae by resection or anastomosis of bowel, &c., even when they have been one or more years in existence, such operations being as a rule unsuccessful and often fatal, a statement which will I feel sure be corroborated by all surgeons who have seen anything of the results of such cases.

Removal of the appendix having been decided upon, a point of some interest is the method which seems most useful for the purpose. I always open the abdomen through the semilunar line; the smaller the incision the better—three inches, or at the most four inches, will generally suffice for dealing with the most difficult case. This, however, judging from the scars I have sometimes seen resulting from operations by others, is not the opinion or experience of all surgeons. Personally I never “deliver” the caecum and appendix in the manner depicted in some of the books unless it is absolutely necessary, because I am sure that the less the caecum is dragged out of the belly the better it is for the patient. I think I am correct in saying that I have in the course of a large experience found it necessary in only a few cases to draw the caecum completely out through the abdominal wound, as in the great majority of cases it is quite easy to free all adherent structures and remove the appendix without much disturbance of the caecum from its bed—this I regard as an important detail. Of the complications, serious and otherwise, following operation, there is nothing more distressing and sometimes more grave than the abdominal distension apt to ensue in some of these cases after removal of the appendix, and I have noticed that the less the intestine is pulled out and dragged from its normal position, the less is the tendency to

distension afterwards. A moment's consideration is enough to show that this is not remarkable. In the actual removal of the appendix the habits of surgeons differ to some extent. I always ligature the appendix after it has been isolated by a strand of catgut placed around it about half an inch from the caecum. The peritoneum, generally greatly thickened, is divided by a circular cut about half an inch to the distal side of the ligature, the mucous membrane being left intact; the “sleeve” of peritoneum, &c., thus formed is turned back towards the ligature as far as possible, the mucous membrane cut across, and the appendix taken away. The short piece of mucous membrane is then freely scraped with a sharp spoon and treated freely with carbolic solution—1 in 20. The peritoneal and muscular coat is then sewn over the mucous stump with fine silk. The removal of the appendix in this way with a sharp knife or scissors is such a precise and easy matter that it is with surprise that one sometimes reads of ingenious plans for effecting this simple proceeding by the use of the electric cautery or other means involving the use of apparatus which is not likely always to be at hand even if any advantage were derivable from its use.

In cases in which the situation of the disease or its extent makes such a methodical proceeding impracticable, the opening left upon removal of the appendix is closed by ligature only or by stitching. When there is any doubt about the security of the closure thus effected I always use an omental graft or plug, which is readily obtainable. In the operation performed in the quiescent stage to use a drainage-tube is entirely unnecessary. In dealing with the abdominal wound I always use fishgut sutures, transfixing the whole parietes, peritoneum, muscles, and skin. I am sure this is preferable to the three-layer system in favour with some, which certainly produces a weaker scar finally and provides much less firm resistance to any straining, &c., which may occur immediately after the operation. Moreover, the separate suturing of the different layers introduces many foreign bodies in the form of sutures, the bulk of which are buried in the parts—an objectionable thing when it can be avoided, as it is a matter of general experience that these buried sutures, however carefully sterilised, do sometimes cause irritation and come away at remote periods.

Cases are occasionally heard of in which within a few hours after an abdominal operation the bowels protrude through the operation wound; this is impossible if the wound be closed by strong fishgut or silk sutures, each of which transfixes the whole thickness of the parietes. With the superimposed layers of sutures it is, however, different, as under circumstances of great strain neither of the three layers of tissue is invariably strong enough to resist the straining effectually. No more disastrous complication than the protrusion of gut from the giving way of the wound a few hours after operation in an appendix case could happen, unless by chance the surgeon be on the spot and ready to return the gut immediately. I have seen one such case in which it was thought undesirable to return the cæcum, which protruded through the wound in consequence of the sutures giving way; the case of necessity terminated fatally.

The main complications arising after appendix operations are pain, vomiting, and abdominal distension. These troubles may or may not be of great importance. Treated rationally, as a rule they subside without causing more than temporary discomfort; if badly managed they may endanger life. Pain more or less as a rule follows the operation; it usually subsides in from twelve to thirty-six hours, and is better left to itself unless too severe, when an injection of morphia hypodermically is usually effectual. Vomiting and distension are more serious matters. I have just recently operated upon a patient—a strong and healthy man—who vomited persistently for three days; it was the most unmanageable case of the kind I have yet seen. Vomiting of this kind may be in some way due to the operation, or may be dependent only upon the anæsthetic. As a rule, vomiting subsides spontaneously if the patient is kept quiet and allowed to sip small quantities of warm water, rectal feeding being used; sometimes, however, it will not yield until the bowels have acted,—the action must be something more than the mere emptying of the large intestine, hence an enema is as a rule not effectual—the only practical means of obtaining the desired result is to place five grains of calomel on the tongue, which usually acts like a charm.

The most distressing and in some respects the most serious complication short of peritonitis is

abdominal distension. This may come on at any period from a few hours to two days after the operation. As a rule, if it does not supervene during the first twelve hours it is of little consequence. In simple cases a large injection containing rue is unsurpassable as a treatment; turpentine in the place of rue may be used, but it is uncertain in result. In many cases an enema only is insufficient. In passing it may be pardonable perhaps to emphasise the necessity of avoiding the introduction of air with the enema. I have known the symptoms of distension greatly increased by the introduction of air in this way by inexperienced or careless people. The familiar saline purgative treatment (*e. g.* 3ij of sulphate of soda every two hours till the bowels act) is often effectual, but in my experience far less so than the old-fashioned nostrum of ʒvj of castor oil and ten or fifteen drops of laudanum, which I therefore employ. Of course if there is vomiting it is inapplicable, and five grains of calomel upon the tongue are then probably the best means of bringing about the desired end.

In connection with the removal of the appendix in cases of so-called relapsing appendicitis the following very pertinent question is not infrequently raised: Does the operation invariably effect a cure? The answer is, in my experience, Yes, provided that the disease is really of the appendix primarily, and understanding also that the word cure is only held to mean freedom from the occurrence of further attacks of a typical kind. It must, however, be admitted that attacks of constipation with feelings of discomfort about the cæcal region follow from time to time in a certain number of cases, especially if the patient is neglectful in the management of the bowels. At times the constipation is extremely obstinate and difficult to overcome; in one case, for example, which came under my notice the constipation was accompanied by the formation of a large, hard, fæcal mass in the cæcum, which was so difficult to deal with that the question of abdominal section was on the point of arising. In conclusion it may be well to remind you that the healing about the parts involved in the operation is often associated with the formation of adhesions, and that such adhesions may, under great strain, within six months or even more from the time of operation, either themselves give way or lead to tearing

of the adherent bowel. In a case upon which I operated death occurred rapidly from acute peritonitis eight months after the operation in consequence of a strain accompanied by severe abdominal pain whilst riding. Apart from the actual tearing of adhesions or bowel by violent exercise, it is, I think, clear that great exertion or strain *before the adhesions are properly organised and tough* might lead to their stretching so as to form elongated bands, which may subsequently tend to intestinal obstruction. I am almost certain that such was the cause of obstruction by a thin band which nipped the small intestine in a patient upon whom I operated for appendicitis, and who insisted upon ignoring all ordinary precautions the moment the sutures were removed from the abdominal wound. It is therefore obviously prudent to impress upon patients the necessity for moderation in violent exercise for at least one year from the time of the operation.

Immediate Reduction of the Deformity of Pott's Disease both with and without Ether.

Dr. J. E. Goldthwait, of Boston, presented a report embodying his experience in this new field. He said that he had soon been impressed with the facility with which the deformity could be corrected and the difficulty of maintaining what had been gained. He had simplified the treatment by using his rectangular iron frame in the application of the plaster-of-Paris jacket. The person lay on cross-straps with the head and shoulders entirely unsupported, thus making traction on the vertebræ. The frame was provided with a windlass for making additional traction, but it had seldom been necessary to use it. He had employed the treatment even in out-patient practice. He had operated upon only one case with abscess, but this one had done well. If the correction could be effected without force, he thought it preferable frequently to change the jackets, thus gaining a little at each dressing. When the disease was advanced, it could hardly be expected, from the knowledge of its pathology, that the gap would be filled in with bone, and consequently one must look for a considerable return of the deformity.

Medical Record.

A LECTURE ON THE CLINICAL INVESTIGATION OF DISORDERS OF THE NERVOUS SYSTEM.

**Illustrated by a remarkable Case of Dual
Consciousness, a Case exhibiting Trophic
Lesions of the Trifacial Nerve; and others.**

Delivered at the Hospital for Nervous Diseases,
Welbeck Street,

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GENTLEMEN,—In former times, when the treatment of disease consisted of the treatment of its symptoms, the clinical investigation of maladies was a comparatively simple matter. The person who suffered from the "spleen" or the "vapours," or whatever it might be, was treated with the prescribed remedy, with but little investigation as to the cause of his illness, and medical treatment resolved itself into as simple a matter as a "penny in the slot" machine, where, if you want chocolate, you put your penny in one side, if you want matches you put it in the other.

But now-a-days all that, I hope, is changed, and the object of clinical investigation is to ascertain the cause of the patient's symptoms, so that we may direct our treatment to that cause. The clinical investigation of disorders of the nervous system is based upon the same principle as that of any other system, with this exception, that being more complex it is all the more necessary to adopt a rigidly methodical system. There should be three steps in every clinical investigation. First we elicit the patient's leading symptom—his most important symptom, though it may not be the one that he regards as most important. Strange as it may seem, it is in this step that the beginner generally fails, the identification of the patient's most important symptom. Secondly, our object is to elicit the history of his illness by a few well-directed, though not leading, questions. And thirdly, we proceed to the physical examination. Upon the information so based we can then return to the leading symptom, and discuss in our own minds, *seriatim*, the several

causes which may give rise to it. Everything, you see, centres round this leading symptom. The indications of nervous disease may be divided, as in other systems, into subjective symptoms and physical signs. And before proceeding to the physical investigation of nervous disorders we will take one of the commonest subjective symptoms. There are not many—nervousness, insomnia, vertigo, headache, neuralgia, and other pains would very nearly exhaust the list. Here is a patient who complains of pain, a symptom extremely common in this as in every other department of medicine. Now there are four qualities which we should note about every pain; its position, its character, its degree, and its constancy. This patient has a severe pain situated in the region of the fifth or trigeminal nerve, of a darting, shooting character, very severe at times, and paroxysmal in its course. This, then, is her leading symptom. Proceeding to the next step, the history of the illness, we learn that three and a half years ago she was laid up for three months with an extremely severe pain in this region, attended by swelling of the face and "ulceration" of the eye, for which she was leeches and blistered. Ever since then she has had now and then attacks of pain, though of less severe degree. On making an examination of the cause you will observe that the left half of the face is flatter than the right, that the skin is atrophied and shiny (glossy skin). She can hardly see with her left eye on account of the opacity of the media; there are adhesions of the iris, and the media are so opaque that we cannot see the fundus. She has evidently had panophthalmitis in that eye. These are the leading facts elicited in this way. Now comes the question, "What are the causes of neuralgia of the trifacial?" The causes of neuralgia of the trifacial and of every other pain may be grouped under three headings: first, we should examine the locality, both the nerve itself and the parts around, for any *local* cause; secondly, we should examine the whole body for any source of *reflex* irritation; thirdly, we should make careful inquiry as to the existence of any constitutional condition. The local examination I have given you. There is nothing to be made out now about the nerve-trunk, it is too deeply situated. As to *reflex* causes, neuralgia of the fifth is very often due to dental conditions, and even when nothing can be made out about the

teeth, hypersensitiveness of the teeth to hot and cold liquids alternately will often elicit an irritated or inflamed pulp. We cannot say at this distance of time whether this cause was in operation then, but the teeth are said to have been falling out about that time (possibly trophic as a *consequence* of the trifacial irritation).

Among the *constitutional* causes might be mentioned acquired syphilis, ague, rheumatism, gout, general debility from any cause, and the like. I have been unable to discover any of these in operation until to-day, but to-day she shows me a tense swelling situated over the inner end of the clavicle; it is obviously a syphilitic node. Here, then, is the probable cause of her troubles. I am glad to have the opportunity of showing you this case, because it illustrates some very severe trophic lesions consequent on a neuralgia of the trifacial; and although the books hesitate to ascribe neuralgia of the trifacial to inflammation of the nerve, there is no doubt whatever that this patient, at the time of that severe attack, had an intense inflammation either of the Gasserian ganglion or the part of the nerve in front of it, probably of syphilitic origin, for the trophic lesions which she has are precisely those which can be produced by an artificially induced inflammation of a nerve trunk or of its ganglion of origin.

Now let us turn to the physical investigation of disorders of the nervous system which present some leading physical sign. I have handed round for your inspection a clinical classification of diseases of the nervous system, and you will observe that all such symptoms referable to the nervous system may be brought under one of the four headings—disorders of muscular action, disorders of sensation, disorders of the special senses, and disorders of consciousness and of the mind. In the first place disorders of muscular action, which may be modified in one of four ways: (1) a diminution of power (paralysis); (2) increase of power, which consists of spasm of two kinds, clonic spasm or tremor, and tonic or continuous spasm; (3) perverted muscular action, such as ataxy, and of equilibrium; and (4) muscular wasting. Before proceeding to investigate some patients suffering from muscular disorders, I would like to remind you that the motor tract consists of a multitude of neurons arranged on an upper and lower level, the upper series being super-imposed upon and

connected with the lower. Here is a diagram representing the condition of things. The upper neuron commences with a brain cell on the surface of the cortex which has a series of processes, most of which are branched and are called dendrons; but one thicker than the rest is not branched, and becomes covered with a myelin sheath; this process is called the axis-cylinder process, or axon. It stretches downwards through the internal capsule, and terminates either in the nucleus of origin of a cranial nerve or else in a ganglion cell in the grey matter of the cord, where it ends in a fimbriated extremity, the fimbriae being not connected with, but close beside the processes from a motor cell of origin, which constitutes the commencement of the lower neuron. The lower neuron commences here in a nerve-cell, and its axis-cylinder process passes through a nerve and terminates in the muscle. You will observe, therefore, that these two series are intimately connected, but not organically so, and that a nerve impression, of whatever nature it may be, is transmitted from one to the other, not by direct organic continuity of the two, but by a process similar to electrical induction. Destructive or paralytic lesions of any part of the upper neuron cause (1) a paralysis, which tends to become spastic, (2) increases the deep reflexes, and (3) is unattended by wasting or electrical changes. But, on the other hand, lesions of any part of the lower neuron produce (1) a paralysis which is from the outset flaccid, (2) which diminishes the deep reflexes, and (3) is accompanied by atrophy and the electrical reaction of degeneration.

Ordinary hemiplegia may be taken as a type of the first, and infantile paralysis as a type of the second. Although the limbs in infantile paralysis tend to become rigid later on, the rigidity is due not to the paralysis but to the contracture of the remaining part of the healthy muscles.

The patient who is now coming in, F. A. W—, is aged 33, and he came complaining to me of weakness of the legs. Had I accepted that statement without careful investigation I should have sought the cause of his disease among the causes of paraplegia. But you will observe as he walks from side to side of the platform that it is not weakness but a disordered or perverted movement from which he really suffers. Turning, then, to the section dealing with perverted muscular action,

you will observe that there are practically only two kinds of perverted muscular action, one is inco-ordination or ataxy, and the other is reeling, due to loss of equilibrium. Now, obviously his affection is inco-ordination, the chief causes of which are *tabes dorsalis*, *Friedreich's hereditary ataxy*, and thirdly any lesion involving the posterior columns. Now, upon investigating the patient we find that his illness had dated from two years ago, when he first had pains in the stomach, with a feeling of constriction round the waist, attacks of vomiting, and paroxysms of pain shooting up the leg from the sole of the foot. He also complained of not being able to feel with the soles of his feet. It was only last March, quite suddenly during a severe paroxysm of vomiting and pain, that he first noticed the legs to be what he calls "weak," but which, as you see, are certainly not weak. It is, in other words, a typical case of *tabes dorsalis*, and you will observe that he has the other symptoms of the affection—*Argyll Robertson pupils* and *Romberg's sign*, namely that he cannot stand with his eyes shut. This last-named symptom has had considerable light thrown upon it lately by the researches of Batten, who has devoted his attention to the muscle spindles, little structures which were known to physiologists but whose function was unknown. They contain the terminations of nerve-fibres, and in process of degeneration these terminal nerve-fibrils always degenerate upwards towards the brain. From this we infer that muscle spindles and their connected nerve-fibrils conduct centripetal impulses. Obviously, then, these nerve-fibrils which terminate in the muscle spindle convey muscle sense, or the sense of a state of contraction of the muscle. Now the means by which a person maintains his equilibrium are chiefly three: first by a knowledge of the state of contraction of his muscles; secondly, by the contact of his skin with the parts around, and especially in the case of the soles of the feet if he is standing; thirdly, by means of vision of the objects around. Now locomotor ataxy is in its pathology a disorder of the sensory nerves. It is always these which suffer first, and Batten has shown that the very beginning is in the muscle spindle, which shows a degeneration. Consequently a patient is unable to equilibrate by this means, nor can he equilibrate by the second means if the soles of his feet are numb; he is

therefore entirely dependent upon his view of objects around, and if he closes his eyes this is also lost to him, and, as you see in this case, he tends to fall.

The next case I want briefly to show you is that of a man who gave me some difficulty in eliciting his leading symptom. He is a man, F. J—, aged 39, a clinical thermometer maker, who has to employ movements of great delicacy, and he complained that his hands shook. But when he came to me they were not shaking, nor did he seem to be ill in any way. But when he was told to pick up a pin or to hold out his hands, then they began to shake as you observe. In other words, he has a very typical "intentional" tremor of the muscles, that is to say, a tremor which only comes on during muscular action. Upon further inquiry this man is found to have several of the typical symptoms of disseminated sclerosis, namely, vertigo, nystagmus, &c., commencing syllabic speech and vacant aspect, but we must not stay to further examine him.

The third case is one which has a very definite leading symptom, namely, paresis of the entire left upper extremity,—a brachial monoplegia. She is a young woman, Frances M—, aged 36, and upon inquiring about the history of the illness we find that she had for ten or eleven years been troubled with some enlarged glands in the neck, which had got bigger from time to time, and at the times when they enlarged she felt particularly unwell. She had also had rheumatism three years ago. Ten weeks ago she was suddenly attacked in the street with paralysis of the left arm and leg, without loss of consciousness. She walked home, taking one hour to do a journey of ten minutes. Thereafter the leg became gradually better, and it was quite well in three weeks' time, but the arm has remained paralysed; it is as a matter of fact getting worse.

Proceeding to the physical examination, we find that a certain amount of power is left in the arm, that sensation is almost entirely abolished up to the shoulder, that it is practically almost flaccid, and that the muscles are already wasting—three features which show us that the lower neuron is affected. Sensation is almost abolished in the whole of the arm, and there is a swelling in the posterior triangle of the neck. As to the electrical changes, there is marked diminution both to

galvanism and to faradism in this arm, with the usual reactions of degeneration.

Turning to the causes of monoplegia, you will see that the chief ones are focal cortical lesions, hysteria, anterior lesions of the cornua, neuritis, and injuries of or pressure on the plexus.

Now the first of these causes, which is rare generally, in the case of the arm involves the face as well. Against the second are the age, the fact that the other arm is quite well, and that all the muscles are equally involved in this paralysis. Against the third, neuritis, which has many things in its favour, is the fact that although it involves all the nerves of the arm, it does not involve any other nerves in the body.

Turning, then, to the last cause, we find in favour of injury to or pressure on the plexus—first, that the whole arm is involved; secondly, that the paralysis is incomplete; thirdly, that there is already commencing atrophy; and fourthly, that *sensation* is equally involved with motion. Finally there is the existence of the presence of the tumour to be made out. She is the subject of lymphadenomatous enlargement of glands, and these have pressed upon the cervical plexus. It is a case for the surgeon, and we shall seek the aid of my surgical colleague.

Next let us turn to sensation. You will observe that sensation may be modified either to touch, to sense of locality, to pain, or to temperature. Now this patient, F. M—, presents a very interesting condition, for she not only has anæsthesia of the arm, but she has hyperalgesia over the areas which correspond to the first, second, and third dorsal spinal segments. The limits of this can be very accurately made out by the rounded head of a pin, which when it touches her she says feels like a tender bruise—in other words, simple touch produces pain. What is the cause of this limited hyperalgesia? Now it has been demonstrated through the researches of Head, Mackenzie, and Thorburn that in visceral disease certain painful and tender areas can be delineated upon the body corresponding to the several spinal segments, and corresponding also to the several visceral diseases. Now this patient has got an area of tenderness over the areas which are reflexly tender in cases of pulmonary disease, and upon examining the thorax she has not only a mediastinal tumour (lymphadenoma), but also dulness at the apex of

the left lung. Here is the reflex cause of the tenderness from which she suffers.

The disorders of the *special senses* and *cranial nerves* is a very big subject, and includes at least two so-called specialities, vision and hearing. It will not be possible for us to go into these large and important branches of neurology to-day.

Let us turn, in conclusion, to that interesting department of neurology, disorders of consciousness and of the mind. Here comes one of the greatest difficulties of clinical classification, for you know how little we are acquainted with the workings of the human mind. I have attempted to arrange these disorders under five groups :

1. Loss of consciousness, which may be partial or complete.

2. Partial loss of the faculties of the mind, which may include (1) lack of attention, such as we all of us get sometimes from overwork, but which may also be due to neurasthenia and general debility from any cause ; (2) lack of memory also comes under this group, such as normally occurs in the aged, in which case it is characterised by loss of memory for recent events only. This also may appear in neurasthenia, overwork, anæmia, and other debilitating conditions. Much valuable psychological work has been done of late years by the study of the memory, and I shall hope to show you directly two interesting cases bearing on the subject.

3. The third group consists of deficiency of mental capacity as a whole, and it consists chiefly of three classes : (1) idiocy (congenital) ; (2) weakmindedness, either congenital or acquired, and imbecility ; (3) dementia, which may be alcoholic or senile in origin, or part of general paralysis, or primary.

4. Consists of perversion of the mental faculties (true insanity). This consists of three or four groups, such as (1) hypochondriasis, which may be defined as an introspective habit of mind which persistently believes the sufferer is afflicted by one or more grievous bodily ailments without cause, but is always desirous of cure ; (2) melancholia, which consists of great mental depression with a strong tendency to self-consciousness and self-negation, without hope, and with a marked suicidal inclination ; (3) emotions or hysterical insanity ; (4) delusional insanity, a delusion being a false idea or belief, an illusion being a false perception

of an actual percept, a hallucination being a *creation* of a false percept. You will observe that I have not made a group for moral insanity ; many of such cases may more probably be included under class 3, and for the rest I hold with Esquirol that "moral alienation is but the first step to all madness."

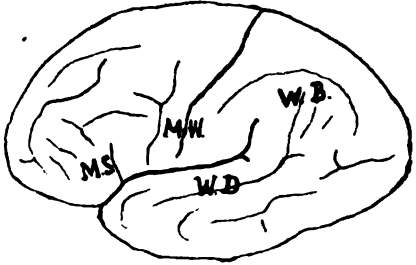
5. Exaltation of the mental faculties may consist either of (1) mania (acute or chronic), or (2) delirium, with which latter we, as physicians, are more concerned.

The patient who is just coming in is a man named E. K—, æt. 41. He was under my care a year or two back for scrivener's palsy, from which he recovered by the use of galvanism, bromides, and iodides, and has remained well until lately. A fortnight ago he was suddenly seized with giddiness, whereupon his mind, he says, became a blank, and he suddenly lost his vision on the right side of both his eyes. Since then he complains that he gets mixed up in doing figures, and that he has completely lost his memory. The hearing, moreover, which has never been very good since he contracted syphilis some years ago, has been worse. I show you a diagram of his fields of vision, which have been carefully taken by my colleague Mr. Work Dodd. You will observe that he has right homonymous hemianopsia, indicating damage to the right optic tract, or else, which is more probable, to the right occipital lobe. But his loss of memory troubles him most, and undoubtedly it is a serious matter, for it is not loss of memory merely for forms or for colours, or for words written or spoken, but for everything. He has probably got some scattered syphilitic lesion involving the cortex.

The next case to which I wish to direct your attention is one of considerable interest, as bearing upon the psychological analysis of the faculty of memory. Here is a table to which I will ask your attention. Thinking persons have long recognised that some people remember better what they see, and others remember better what they hear. The former can commit things to memory better by reading, the latter by being read to or hearing spoken words.

I saw a patient æt. 24 in consultation some months ago, and she has been kind enough to come and see me to-day. Her history is as follows :—She has always been regarded as a

PSYCHOLOGY OF MEMORY.

PSYCHICAL CONSTITUENTS OF MEMORY.	EFFECTS WHEN LOST.	POSITION IN BRAIN.
Auditive image	Non-recognition of spoken words (verbal auditory amnesia), or formerly "word deafness"—W.D.	
Visual image	Non-recognition of written or printed signs (verbal visual amnesia), or "word blindness"—W.B. Non-recognition of forms { objects, persons, and places. " colours	
Motor { Image of articulation Image of writing	Loss of intelligible speech —M.S. Loss of intelligible writing —M.W.	

delicate girl, but with nothing serious the matter. In January, 1896, she had a very serious quarrel with her sister about a young man, to whom it was suddenly apparent they were both attached. It preyed upon her mind very greatly, and she hardly spoke for a couple of days, during which time she went to see *The Sign of the Cross*, a play which evidently had a very marked emotional effect upon her. The next day she had a very violent attack of hysterics, and thereupon became cataleptic. This condition lasted for several weeks.

names. By degrees, however, at the end of three or four months she recognised her mother and the doctor, but still called her elder brother the big boy, and her younger brother the little boy. Finally she recognised these. Her memory for objects, forms, and colours then returned first. By degrees her commemorative auditive image returned, and she recognised words which were spoken to her. Her motor image for articulation was re-established, and finally, but not until the expiration of nearly twelve months, her motor mental image for articu-

Dr SAVILL

Dr SAVILL
Dr SAVILL

She was in a state of semi-somnolence, had to be fed forcibly, took no notice of any object or person around her, and passed her evacuations under her. It was not till many weeks had elapsed that she began to talk, and then she talked garrulously and unintelligibly, not recognising any person or thing around her at all, calling them always by wrong

lation was partially restored, and she was able to speak intelligibly; but she still calls things by wrong names, e.g. any vehicle she calls a chariot.

At the present time she is still, even after such a long period, unable to read and unable to write. Her commemorative visual image of written and printed signs, and the motor mental image for

writing, is still unrestored; she can neither read nor write. And though, as you see, I endeavour to get her to write or to read, she is unable to do either. I thought perhaps she might be able to copy letters in the way children are first taught to write. I show you here her attempt to copy from the bold letters in which I have written my name. The copy is like that of a little child just learning to write, and she took nearly ten minutes to do it.

It is extremely interesting, then, to note in her the gradual re-establishment of the different psychological divisions into which memory may be divided. There is, however, another and perhaps a still more remarkable point about this case. She lost in January, 1896, as we have seen, practically the entire faculty of memory, and by degrees its different parts have become re-established. In her youth she strongly disliked needlework, and was very clumsy with her needle. Since the attack in 1896 she has suddenly, and of her own accord, taken very much to needlework, and has equally suddenly developed into a most skilful needlewoman, executing the most difficult and elegant pieces of work imaginable, a talent which I am told generally takes some considerable time to acquire.

As regards the treatment of this case, there are three ways in which these last faculties of the memory and of reading and writing can be restored. First by educating them in the same way as we do children, setting them to draw pot-hooks and to copy letters. Secondly, they often learn of their own accord to substitute one form of memory for another; and there is a very remarkable instance of this mentioned by Charcot in his lectures,* of a merchant who suddenly lost his mental vision of signs and objects, but who gradually "realised little by little that he could by other means, by invoking the aid of other forms of memory, continue to successfully direct his business affairs." Thirdly, there is another means which I hope to be able to adopt in this case, and that is the aid of hypnotism to try the efficacy of the suggestion of ideas in a hypnotic state. I propose to invoke the aid of my friend Dr. Lloyd Tuckey; and if

she is hypnotisable we hope to be able to do something to restore the lost faculties of her mind.*

NOTES, &c.

Infantile Diarrhoea.—Dessau is of the opinion that in an acute attack of summer diarrhoea in a child under two years of age all albuminous and starchy foods should be withheld at once. Instead, toast water, made by laying in a large bowl two pieces of stale white bread toasted brown on both sides, pouring on boiling water till covered, adding a pinch of salt and allowing to stand till cool, the clear water being then poured off into a fruit-jar and kept cool by ice, is excellent. Barley water, made by boiling a handful of pearl barley in a pint of water for one hour or more, a pinch of salt being added, can also be prepared, and after it is cool the supernatant liquid poured off for use. From one to three tablespoonfuls of either of these foods can be given every hour or two for forty-eight hours if necessary. Alcoholic stimulants may be added if necessary. These drinks should always be given cold. When vomiting and stools have improved, which usually occurs within forty-eight hours, nursing may be resumed at intervals of either two, three, or four hours. If sterilized milk be used it should not be for longer than the summer months, on account of the tendency to produce rachitis. A mixture of cow's milk, diluted one fourth with water and containing a little milk-sugar and a pinch of salt, is to be preferred. The prepared milk is poured into a

* Charcot's Clinical Lectures on Diseases of the Nervous System, New Sydenham Society's translation, Series iii, p. 154.

* The visit to the hospital, and the re-establishment of the menstrual function, which had been absent six months, determined in her a fresh attack of hysteria, followed by catalepsy lasting some twenty-four hours. On recovery from this her whole manner was changed; she lost the whining affected voice, and regained her former manner, and at the same time regained her memory for written or spoken words and her power of writing, and it was found that she had completely abandoned the false nomenclature of her abnormal self. In a word, the case is one of dual consciousness, the second or abnormal state of mind and memory being induced by the first prolonged cataleptic attack; her normal mental condition being restored by a second, though milder attack.

double boiler of agate ware, and the water in the outer vessel is allowed to boil for fifteen minutes. The inner vessel is then rapidly cooled, and the contents poured into a well-scalded tight fruit-jar, and kept by the ice until required for use. The entire quantity required for use during the day can thus be prepared at once. After each feeding, the child's mouth should be wiped out with a bit of absorbent cotton soaked in a saturated solution of boric acid. Plenty of water that has been boiled and cooled should be given.

Gilbert contends that milk should be excluded for at least three days. The white of a fresh egg beaten up with crushed ice and a pinch of salt is most acceptable to the stomach and is sufficient nourishment. Pure sterilized water may be given freely. Soups, meat broths, and starchy mixtures should be rigidly excluded. A most potent adjunct in the management of protracted summer diarrhoea is intestinal irrigation with the normal salt solution, one drachm of sodium chloride to the pint of warm water. With a No. 18 soft rubber catheter attached to the nozzle of a fountain syringe filled with the salt solution, one can easily distend the colon. The hips of the child should be slightly elevated. The irrigation should be continued until the water returns clear and free from fecal matter.

Epstein, of Germany, in the summer of last year practised the subcutaneous injection of a salt solution in acute digestive disorders and cholera infantum. He reports prompt improvement and quick cures in cases that were apparently hopeless. Epstein used $2\frac{1}{2}$ drachms of the normal salt solution at a time hypodermically.

Robinson makes a plea for the more general employment of intestinal irrigation and rectal alimentation in the grave diarrhoeas of children, giving the result obtained in one of his cases. Child 8 months old, suffering from diarrhoea; temperature 106.6° F.; pulse impossible to count; respiration very shallow and feeble. Irrigation was followed by the discharge of large amount of feces, over two gallons of starch water being used before the return water came out clear. The child was then put into a hot mustard bath, cold water being poured from a distance of about three feet on his head and forehead. The bath lasted six minutes, after which the following mixture well beaten up was injected into the rectum: 1 egg,

1 tablespoonful of cream, $\frac{1}{2}$ teaspoonful of brandy, teaspoonful of beef juice, and a teaspoonful of a mixture containing potassium bromide 3 grains, chloral hydrate 1 grain, antipyrin 1 grain, and mint-water 1 drachm. The buttocks were pressed together for about five minutes; the child put to bed with hot-water bottles to his feet and cold-water compresses to his head, and he immediately fell asleep. Within three-quarters of an hour the temperature fell from 106.6° to 102.5° F. The child at the end of a week was perfectly well. The rectal alimentation was continued with slight changes every four hours for four days, then peptonised milk was carefully tried by mouth.

During the hot weather of last summer Bowles treated between sixty and seventy cases of summer diarrhoea in children ranging in age from a few weeks to three years. The cases were in every way such as are met with in the crowded tenements of large cities during the heated term. He used lactic acid in every case. The maximum dose was $1\frac{1}{2}$ grains given every hour. The result was the disappearance of all symptoms in from twenty-four to forty-eight hours. The only medicine given beside the lactic acid was an initial dose of calomel in cases where it was indicated.

Neville has found nothing so valuable as listerine, combined with chalk mixture or bismuth, in the summer diseases of children.

Comby has given tannin and its derivatives, tannigen and tannalbin, a prolonged trial in the treatment of infantile diarrhoea. Powdered tannin is unsatisfactory. Tannigen and tannalbin are compounds which are broken up in the intestine with the liberation of nascent tannin; this has the advantage that the stomach escapes the astringent action of pure tannin. He has given tannigen and tannalbin with very satisfactory results in the simple non-infective diarrhoea of children. They are not adapted for cases of cholera infantum. They may be given for days or weeks without untoward results in quantities of 8 grains to 23 grains in the day, according to the age of the child, quantity being divided into three or four doses.

Fenwick uses resorcin, having found it highly satisfactory in a large number of cases of intestinal dyspepsia in infants and young children.

Mikhnevitch, having tried the salicylate of bismuth in fifty cases of diarrhoea in infants under

two years of age, reports that only two died. The following formula is recommended :

℞ Bismuthi salicylici	24 grs.
Gummi arabici...	1 dr.
Sacch. albi	1½ drs.
Terendo adde aq. dest.	2 oz.
Fiat lac, tum adde aq. dest.	4 "

M. D. S. The bottle to be kept in cold water or ice, and to be shaken well before use. One or two teaspoonfuls three to six times daily. In cases of offensive diarrhoea the administration should be preceded by a dose of castor oil. In acute cases the remedy is useless, but in all of a week's standing or longer its effects are excellent.

Tompkins speaks highly of the following as an intestinal antiseptic in children :

℞ Calomel...	2 grs.
Sulphocarbolate of zinc	3 "
Subnitrate of bismuth	2 drs.
Pepsin	½ dr.

Sufficient for twelve powders. Three per diem in a child of one year.

Crandall presents, as follows, the indications and contra-indications for the use of opium in the diarrhoeas of young children. It is contra-indicated:—(1) In the first stages of acute diarrhoea, before the intestinal canal has been freed from decomposing matter. (2) When the passages are infrequent and of bad odour. (3) When there is a high temperature or cerebral symptoms are present. (4) When its use is followed by elevation of temperature, or the passages become more offensive. It is indicated:—(1) When the passages are frequent, with pain. (2) When the passages are large and watery. (3) In dysenteric diarrhoea, together with castor oil or a saline. (4) In late stages, with small, frequent, nagging passages. (5) When the passages consist largely of undigested food, and the bowels act as soon as food is taken into the stomach.—*Monthly Cyclopædia.*

Appendicitis complicating the Pregnant and Puerperal States. By S. Marx, M.D.—One of the most interesting conditions complicating the gravid state is the inflammatory process in the right iliac region, a pathological factor engrafted upon a physiological condition. At best the appendical inflammation is rare during pregnancy, but I believe appendicitis occurs with greater frequency than we have heretofore been

taught to believe. To my mind there is no valid reason why appendicitis should not occur more frequently during pregnancy and the puerperal period than under other conditions. When we consider the frequency of appendical inflammation in this country among males, and with somewhat less frequency in females, we have here a marked predisposition. Further, during pregnancy the enormous congestion of the entire vulvo-vagino-uterine tract, which is readily reflected upon the entire intestinal system, causing as it does plethora followed by torpor of the gut, with the subsequent marked constipation—we have here plainly an exciting cause which becomes almost clear evidence ; with this the generally depraved and vicious state of blood will gradually fill in the circle of predispositions. During the parturient act direct traumatism applied to an unusually long appendix, prolapsed over the brim of the pelvis and open to the prolonged, well-directed, and continuous pressure by some surface of the advancing foetus, will readily give rise to an acute inflammation of that organ. It is to the diagnosis that I wish to call special attention, for here we meet the first great stumblingblock. This in most cases, from personal experience, is difficult and perplexing, for we must always remember the high situation of not only tube and ovary, but the entire broad ligament and uterus. The mere fact of finding the right vaginal fornix free from fulness, exudate, or other evidence of an acute inflammation, does not by any means exclude an acute inflammation of the appendages ; and, on the other hand, the presence of such evidence in that neighbourhood, while strongly presumptive of tubo-ovarian disease, cannot positively imply that an appendicitis is absent. It is only by carefully studying both conditions that a diagnosis *may* be arrived at, and in a few cases a differentiation cannot be made. But hair-splitting differences cannot have an important bearing upon the case, since it is only a "stickler" in diagnoses who will advance arguments *pro* and *con*, and to what end? We can gain nothing, for if the condition of the patient is such that an operation seems necessary, it will make absolutely no difference in the prognosis, present and remote, as to whether the pus to be evacuated or the organ to be amputated be either appendix or tube and ovary. Thus I well remember two cases in which an appendicitis post partum was diagnosed. In the

first case a clear history of appendicitis which was recurrent was given the physicians in attendance. The location of the tumour, McBurney's point, and every feature was typical. The operation, performed by a distinguished surgeon two weeks after a precipitous labour, revealed a tubovarian abscess of long standing. In the second case, four days after a premature delivery I saw the case in consultation. There was no evidence of uterine sepsis, the young woman had had a chill right after labour; there were present the severe abdominal pains and the rigidity of the right rectus muscle; the temperature was not high, the pulse rapid; there was constant vomit. A well-marked tumour in the right appendix region and the absence of signs of inflammation in the right vaginal vault justified a diagnosis of appendicitis. The patient was removed to a hospital, and an operation performed by an incision in the right iliac region. A dense cellulitic mass was discovered, involving the entire right pelvic region. No appendicitis found, and no pus present. Post-operative diagnosis, right-sided pelvic cellulitis in an unusually high situation.

My experience in the surgical use of serum is rather limited, and yet from my small number of cases (purely surgical) there is a decided suspicion that in the streptococcus antitoxin we have a very valuable adjuvant to our therapeutics which ought never to be omitted. In obstetric streptococcus cases my experience has been large, and I have yet to save my first case by its use. These cases do not include cases of pure sapræmia, where there is merely a toxic absorption, in which the curette, douching, and thorough drainage are all that is necessary for a prompt and complete recovery; but in my list are the pure or mixed streptococcus cases, characterised practically by absence of all local symptoms and by the presence of low temperature, high pulse, and the presence of the micrococcus pyogenes in the blood. In these cases, as was above stated, the serum has always failed; and why? I do not know. There must be some modifying influence which the puerperal period exercises over the toxins thrown into the circulation. As to the dosage of the serum, while I have always used large and repeated doses, as well as the smaller amounts, I cannot see that the therapeutic effects are any different. From a personal experience there have been noted unpleasant

symptoms from single large doses which were not observed when smaller amounts were used, and in some cases the symptoms presented were such as to warrant me in surmising an acute poisoning from the serum; intense headache, nausea, and decided and repeated vomiting, with very rapid pulse, high temperature running up 3° to 5° within a short time after exhibiting the drug; this condition being followed by profuse sweating and prostration, leaving the patient, if anything, worse than before. The good effects noted by the drug, and where it seems to be doing favourable work, is either a fall of temperature at once or a temporary slight rise, decided falling of the pulse, profuse diuresis, and a not exhausting diaphoresis. Under these conditions it can be safely administered. On the other hand, where persistent high temperature, rapid pulse, profuse vomit occur, the drug is doing harm. Note, for instance, the following case, where a rather mild septicopyæmia followed a total hysterectomy. Micrococcus pyogenes demonstrated in the blood; temperature never over 101° ; pulse about the highest 120° ; serum used, 10 cubic centimetres every twelve hours. At once temperature 103° to 104° , pulse in proportion, incessant vomiting, great prostration, &c. The serum continued in smaller doses every six hours, but the high temperature and rapid pulse, without variation, are present. Suspecting the serum to be at the bottom of the trouble, the hypodermatics of the same are omitted, and in twelve hours the condition changes at once and convalescence is fully established. So marked was the change in the condition of the patient that it was evident to everybody connected with the case that the serum was causing the disturbance. — *American Journal of Obstetrics and Diseases of Women and Children*, August, 1898.

APPEARANCE in the blood-serum, under the influence of chemical products, of a substance capable of agglutinating the bacillus of tuberculosis. — Arloing causes the appearance in the blood-serum of goats of a principle agglutinating tubercle bacilli by repeated injections of eucalyptol, guaiacol, creosote, or Mialhe's solution (corrosive sublimate). As none of these substances has any agglutinating power outside of the body, it must be assumed that they produce in the body certain chemical substances of which they themselves are devoid.

La Semaine Médicale.

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WITH

PROF. SIR T. GRAINGER STEWART IN THE WARDS OF THE ROYAL INFIRMARY, EDINBURGH,

June 22nd, 1898.

MR. WILSON, clinical clerk, read the notes of the following case:—A man æt. 60 came complaining of an uneasy feeling about the stomach, which had lasted three weeks. Hereditary tendencies had no bearing on the case. His habits as to food and drink were good, and he was very temperate. He smoked about two ounces of black twist per week. His occupation was extremely healthy, as he had plenty of outdoor exercise.

Previous illnesses.—As far as the patient remembers he never was ill until nine years ago. At that time he first suffered from indigestion, with occasional vomiting. He was treated at home from time to time by his own doctor. After that, as he did not improve, he was recommended to come into Ward 22. The patient was unaware that he was bloodless until he came into the infirmary. He was in the ward on the first occasion for twelve weeks, and was treated for pernicious anæmia. After that he went home again, and resumed his work. Since then he has had three relapses, for each of which he was treated in the same ward, the last three and a half years ago. On each of these occasions the initial symptoms were those of indigestion and vomiting. This was followed by weakness and shortness of breath, but no pain. The patient has been working for the last three and a half years.

History of present attack.—Three weeks ago the patient noticed that he had uneasiness in the region of the stomach, which came on after food, usually about ten minutes afterwards, and was occasionally accompanied by vomiting. The vomited matter did not taste badly. No history of blood in the vomit. The patient vomited on an average three times a week, his appetite was very poor, and he

often ate very little. He noticed that broth and potatoes upset him most, so he lived chiefly on milk puddings. He did not have a doctor, but in three weeks' time came to the infirmary. The shortness of breath during the last three weeks has been more marked than usual. The bowels have been quite regular.

State on admission.—Present weight 11 st. 5½ lbs. His weight six months ago was 12 st. Development and muscularity good. Conjunctivæ are faintly tinged with yellow. The skin has a sallow and ashen appearance (this occurs during every attack). General appearance of expression of face contented. Temp. 99.4°.

Alimentary system.—Lips pale, gums pale. Tongue furred. No difficulty in swallowing. Appetite poor. No special thirst; no special sensations during fasting or eating. He complains of a pain coming on about ten minutes after food, lasting about one hour. No feeling of weight, but a feeling of distension of the stomach. No heartburn or nausea; no acidity, but flatulence and water-brash. The patient vomits on an average once every second day. This vomiting occurs sometimes before sitting down to a meal. The vomit is chiefly sticky glairy mucus, small in quantity, about two mouthfuls, and difficult to get up. No sour taste or smell.

Abdomen.—On inspection there is nothing abnormal. Palpation reveals nothing. Percussion is normal.

Liver not enlarged. Stomach not enlarged.

Hæmopoietic system.—No enlarged glands. Spleen not enlarged, not extending further forwards than the mid-axillary line.

Blood.—Red blood-corpuscles 800,000, leucocytes 3600, hæmoglobin 36 per cent. Films show marked poikilocytosis and no nucleated red blood-corpuscles.

Circulatory system.—No pain or faintness; marked dyspnoea during attacks of illness, more marked on exertion. Pulse: vessel wall normal; character, frequency 72 per minute, regular, feeble; tension between beats low.

Inspection.—Apex-beat not visible. Palpation: apex-beat feeble in fifth space five inches from middle line. Percussion: right border of heart one and a quarter inches from middle line, upper border at second interspace, left border five and three quarter inches from middle line.

Auscultation.—Soft blowing systolic murmur in mitral and aortic areas. Heart-sounds faint. Second sound reduplicated in tricuspid area.

Respiratory system.—Breathing twenty per minute, regular in rhythm, type abdomino-thoracic; no pain; no cough; no spit; no sign of hæmorrhage in mucous membrane of pharynx or larynx. Inspection normal. Palpation: fremitus slightly increased at right apex. Percussion normal. Auscultation. Duration of expiration prolonged, especially at right apex anteriorly. Type harsh, vesicular. Vocal resonance increased at right apex anteriorly in second space. Accompaniments: crepitation at both bases, chiefly dry and high pitched.

Integumentary system.—No subjective phenomena. Signs of subcutaneous hæmorrhages in various parts of the body.

Urinary system.—No subjective phenomena. Urine, sp. gr. 1011, acid; no abnormal constituent; no deposit. Reproductive system normal.

Nervous system.—Sensory functions: no subjective sensations. Sensibility to touch, heat, and tickling and pain normal. Muscular sense normal. Pupils react to light and accommodation. Fundus normal. No hæmorrhages. Motor functions normal. Vaso-motor and nutritive functions normal. Cerebral and mental functions normal. Cranium and spine normal; locomotory system normal.

Well, gentlemen, this is a case of very great interest to me in that he has been so long under observation. I told you the other day when he came in that he had been often in the wards for a number of years, and you have heard the particulars of his former visits read. It was in 1890 that he first came in, and was sent by Dr. Skinner, of Lauder, who had been attending him. He says he was ill for a considerable time before he came to us the first time. I gave a clinical lecture upon him in 1894. He came in with pernicious anæmia; he had the lemon tint of skin, which I ask you to carefully note now. It so happened that just at the time that he was here my former pupil and friend, Dr. William Hunter, who has written so much on this subject, went round the wards with me, and he said that this man's case was a perfectly typical one, and I think he said it was the worst case of pernicious anæmia he had seen. In addition to the ordinary symptoms, our patient

showed two which were very distressing, namely, restlessness and sleeplessness. He will remember what hard work it was at one time to keep him in bed; we had to give him various medicines to quiet him and procure sleep. The medicine which did him most good was paraldehyde, but we had to give it in very large doses, on one occasion as much as seven drachms at one time; with the aid of that he was able to get a little sleep and rest. Of course we tried all the ordinary remedies with him, we put him upon iron and arsenic, but they completely failed to afford him any benefit. Then, in despair, we put him upon tincture of perchloride of iron in very large doses, far above the amount which can be absorbed physiologically; but, of course, this is one of the instances in which the physiological work of remedies does not correspond to what you see in disease. Clinically, we saw how large a dose could be tolerated and absorbed by this man, and that he improved under it in a most marvellous way; gradually his blood got better, and at last he was able to return to his duties as steward of a farm. The farm over which he was steward was near a place I rented, and I was one day taking a walk with some friends over the moors, and came upon a piece of cultivated land and got in amongst the harvesters, and on asking where we were I, to my astonishment, was greeted by my friend here, who was looking after the party and actively conducting things, and looking not a bit the worse for his old pernicious anæmia. Of course I had no facilities there for counting his blood-corpuscles, but I looked at his gums and lips, and I could see his vigour, and in all respects he seemed perfectly well, and we see him from time to time in the enjoyment of good health. Every time he has come into the infirmary it has been with a recrudescence of the old trouble. Every time Dr. Hunter and I meet we talk over this case; he is always anticipating recrudescences, and I am always reporting recoveries. Here is another recrudescence which we have to face, and I want those of you who have not seen him before to note that it is a very typical case. Do you believe you can absolutely mark off pernicious anæmia from other kinds? Not in all cases; there is in this as in other departments no definite boundary line—we cannot say where simple anæmia ends and where the pernicious anæmia begins. But if you are going to say that a patient

who does not die of anæmia has the simple form, then, of course, this man having lived for nine years after his first illness has simple anæmia. If, on the other hand, you are going to take the group of symptoms which mark pernicious anæmia, you have them here in a typical form.

Now what are the great features which you value for the diagnosis of pernicious anæmia? Extreme pallor, a lemon tint, great weakness, often vomiting. Then other points are that they do not generally do good under iron, but more frequently under arsenic. Sometimes bone marrow benefits but it turns out that though that is a thing which sometimes does good, its remedial effects are not by any means constant. Then there is a symptom of the skin which is of very great importance, namely, little hæmorrhages are very common indeed.

Now I think it would be best for us to go over the different systems, and bring out the points in our usual order, so that you may gather up the facts with regard to pernicious anæmia accurately; that is to say, those of you to whom the disease is a new one.

In the alimentary system you expect to see pale lips and gums. If you examine the gums you will see that if there is any difference in the colour of the teeth and the gums, the gums are the paler. As to the conjunctivæ, he has little yellow thickenings of the conjunctivæ, and you notice that the skin is pale about the mouth, and so on. Further, with regard to the alimentary system, you, of course, would not expect a man with such a disease to be able to secrete from that blood the material required for digestion purposes. And there are several good reasons for this failure. He may fail because he has such poor blood that he cannot afford the material, or because his glands are in such a bad way that they cannot separate the material and produce the secretion. Again, the nervous system may determine the difficulty. You may have a change of secretion into the stomach on the part of the intestinal glands, because of the deficiency of the blood, or because of the deficiency of the mucous membrane, or because of the deficiency of the innervation of the secreting structures. We have no particular reason to suspect that innervation fails here; we have no reason to believe that he has got catarrh, but of course you should examine the

tongue of such a case; you see it is not badly furred. It is mainly the fault of the blood that he is not able to secrete very satisfactorily. Now as to the condition of the bowel, the patient says his bowels act very well, practically every day, and he further says that he notices no difference in the action of his bowels when he is well and when he is ill; diarrhoea is a condition which he very seldom has, he says. You have heard that the liver is of normal size, therefore we will not now percuss it out.

Our natural step from the alimentary system is the hæmopoietic system. We have seen this poor fellow's blood on many occasions, and the report as to the state of the blood is that on the 14th of June there were 800,000 red corpuscles per cubic millimetre; on the 16th, 835,000; on the 18th, 810,000; on the 19th, 883,000; on the 20th, 845,000; on the 21st, 860,000. The leucocytes were 3600 at first, now they are 4250. On the day that the red corpuscles fell a little, the leucocytes were up to 5400. The percentage of hæmoglobin was 28 per cent. on the first day, it is now 35 per cent. As to the appearance of the corpuscles, perfect examples have been carefully selected and put out for you under the microscope, by which you will be able to judge of the condition. No nucleated red cells have been found. A stained film of normal blood is put alongside by Dr. Chiene for comparison. In addition we have to remember about the hæmorrhages. Next you have to notice whether there are any red spots. You will see that he has hæmorrhages.

Passing from the hæmopoietic system we come to the circulatory system, and the points to remember with regard to that are, at the mitral area and base of the heart is a systolic murmur of a soft blowing character, such as you get in cases of anæmia. With regard to the pulse, as we have been told in the record there is no thickening of the walls of the vessel. The man is in his sixtieth year. The beats are not strong, but quite regular. The tension between the beats is low. We do not find any evidence of organic change here; but the pulse is not strong, and the heart-sounds are very feeble, by which we know that the heart is lacking in vigour and tone. You cannot expect a heart to be very vigorous with blood such as this man has. Such a heart cannot be strong, but it may be tumultuous. Weak hearts you know are

like weak people, they are apt to be a little fussy, and the weaker they are the more fussy they are apt to be. These feeble hearts are often very fussy and excitable, and have a somewhat spurious strength which is very far from being real. Now in this man's case the heart is somewhat dilated. What do you picture to yourself as the condition of the muscular wall? Would you expect to see beautiful transverse lines, or would you be prepared to see some spherical points reflecting the light? You would be prepared for some degree of degenerative change. Therefore one of the things we have to set ourselves to do is to build up the muscular fibre of this heart.

With regard to the respiratory system, Mr. Wilson said it was not very much to complain of. It is a little altered in the physical signs, but on the whole his lungs are sound enough. In every case of this kind your experience has taught you to test the bases of the lungs behind, and in your consulting rooms you try to detect crepitations at the back of the lungs. It is the only kind of thing to which a patient of this sort is commonly liable in enfeebled circulation. There is now not a trace of moisture at either base.

With regard to the integumentary system, we have already referred to these hæmorrhages, and to the general lemon tint of the skin and to the extreme pallor.

Next as to the urinary system; what do you observe about it? There is no abnormality of the urine, and the patient says he has very seldom noticed any change in the colour of his water, but as he generally micturates in the open air he has not many opportunities for observing. Sometimes when there is much weakness the patient passes water of a deep sherry colour.

With regard to the nervous system, Mr. Wilson tells us that he has found this man's nervous system very fairly sound. He is sleeping well at night, has no aches and pains about him, no giddiness, no headache. Neither is there any change in sensation. There are no symptoms of alterations in the cord or other parts which we know to occur in cases of pernicious anæmia. Remember that lesions have been discovered in the spinal cord and also in the brain in the course of pernicious anæmia. But in this case Dr. Chiene has searched and failed to find any token of anything of that sort.

As to the locomotory system, do you, with your mind's eye, see anything the matter with his bones? There are, as you know, two kinds of bone marrow, red marrow and yellow marrow, It is very likely indeed that the red marrow is very deficient, but of course we have no proof of it. Then what about the muscles and joints? You say the muscles seem well developed, but I ask you to feel his biceps, and you will have the impression that the muscles are not now deriving very much nutrition. He tells us that he could not now fight a stag as he could if he were well, notwithstanding his years.

Now, gentlemen, the facts are before you, the outline of pernicious anæmia. You will remember pernicious anæmia cannot with certainty be differentiated from the simpler forms of anæmia, and that beyond a certain line it is practicable and legitimate for us to speak of pernicious anæmia. But, on the other hand, while we have always to be anxious about our pernicious anæmias, we are *never* to give them up. You have to go on treating them, you are to expect them to improve. *Many* a patient have we seen in these wards with a bad pernicious anæmia who has improved and improved, at least for the time. But here is the great leader of them all; he goes on improving every time, and we hope he will do so again. Now we are trying with him a new material which I pass round for your inspection; it is the iron salt of phospho-carnic acid, produced from milk, which is believed to be a very fine blood-forming material. One of the laboratory students in the University is working at the question now, and I am putting all the facilities that I can in his way for testing different remedies in cases of this sort. There has not been time yet to make the least sure, nevertheless we are going to give a trial of them, and we will see how he gets on. If this fails we shall try arsenic and iron with him, and various other things with a view to restore the condition of his circulation, and hope for another rally before very long. Last time he was here he did very well on iron and Flitwick chalybeate water with tincture of perchloride of iron mxx , three times a day; at one time he was having mxx every two hours.

Now I want to devote the short space of time which remains to us to-day, to the unfortunate heart case which has compelled us to interrupt our regular teaching. The poor fellow was ad-

mitted yesterday very ill, sent in by an exceedingly shrewd doctor in the country, who wished him to get the benefit of hospital treatment here. He is in the most extreme danger, and it is doubtful if we shall be able to rescue him from death. But of course we will do the best we can towards that. He has been suffering from chronic valvular disease, with both aortic and mitral murmurs. There is also a little cirrhosis of the kidneys. For a considerable time he responded to digitalis, his doctor says, but it now produces no effect. He has also been tried with regulated doses of strophanthus, with little or no result.

In conclusion, his doctor suggested that perhaps we might keep him in the infirmary for a time, and ease him a little. He came some twenty miles here, and the journey has probably proved too much for him. The state of matters is this, that effusion has taken place into his pleura to some extent; he has got great over-mastery of the heart, and great difficulty in breathing. He has been bled a little while ago, and I doubt if we can repeat the process. But we shall now try to get some fluid out of the thorax. [Ten ounces were then drawn off with an aspirator, and the patient experienced considerable relief. But it proved only temporary, for death took place in the course of the afternoon.]

The Effect of the Mauser Rifle Bullet.—All of the cases tend to confirm previous observations to the effect that the small calibre bullet of the Mauser rifle causes wounds of the soft parts, which, if left alone under the first dressing, will heal by primary intention in the course of a week or two unless complicated by serious visceral injuries. The cases corroborate the statement that the small calibre bullet does not ordinarily inflict a bad wound, and that it seldom carries with it into the tissues clothing or other infectious substances. This observation is an extremely important one for future field service, as it must satisfy field surgeons that such wounds will heal promptly if left alone under the first aid and antiseptic dressing. On the other hand, I have seen the evil consequences following meddlesome surgery in the form of unnecessary probing. Such wounds are susceptible to secondary infection caused by the use of the probe.—N. SENN.

The Corpuscule, September, 1898.

Some Considerations with regard to the Treatment of Pulmonary Tuberculosis, and the Limitations to the Usefulness of Antitoxins and Antiseptics in the Treatment of this Disease.

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THOUGH clinical observation may lack the precision of experimental research, it has a value to the student of disease which cannot be surpassed by any other means of investigation. Experimental research has cleared up much that might otherwise have remained long obscure, and has given wider meaning to much that is observed at the bed-side; indeed, the chief end and use of experiment is to elucidate the clinical signs which it is the province of the physician to appreciate correctly, with a view to effectual treatment. The sum of what may be gained from noting the allied processes occurring in animals only suggests possible explanations of what occurs in the human subject, and these explanations can only be adopted when verified by clinical observation. That this must of necessity be the case is obvious when we consider how differently the same disease may affect different persons; and since the same disease may be modified in different individuals of the same species, it is possible that in animals of another species the same morbid cause may produce quite a different chain of events. It is the recognition of this fact which has led to more careful consideration of the "personal equation" in our observation of disease. We aim at treating the individual who is ill, instead of applying the same remedies to all cases of a given disease. This is the rational outcome of the closer study of pathology and morbid anatomy. From such study it becomes evident that the conditions of organs and tissues are constantly undergoing changes—apart from any definite diseases—as the mere result of life; but these changes differ somewhat in different individuals according to the mode of life. &c.

Even at birth there are certain subtle differences

between individuals—the impress of heredity—which, though not immediately obvious, exert a manifest influence as development proceeds, and stamp their mark on all the subsequent life-changes. Pathologically, persons do not all start in life from the same point; the race of life is a handicap. Even those who are closest together at the start soon diverge, and in a few years their individuality is as certainly stamped on the organs and tissues as it is in face, form, and manner.

Thus a morbid condition attacking the same organ in two persons of the same age would not produce precisely the same pathological picture in that organ in the two cases; the outline might be the same, but there would be differences in the details. Still more would the same disease show modifications in persons of different ages and of different classes or races. But it is not only the "soil" which may cause modification in the manifestations of a morbid agent. The dose and continuity of action of the poison which starts the illness will vary in different cases, and the virulence of the infective material may be subject to variation.

Thus the same disease is sometimes widely different in its course and effects, not only in different animals, but in different individuals. Recognising this fact, treatment ceases to be entirely a question of "specifics."

One of the most noteworthy characteristics of medical progress in recent years has been the recognition of the modifying influence of the individual on the manifestation and course of the disease—what has been termed the personal equation in disease. Almost before this principle has been thoroughly appreciated we are being drawn again towards the search for specifics. Our *fin-de-siècle* therapeutical armamentarium would almost have satisfied the savage medicine man or the astrologer of the Middle Ages. Instead of serpent's blood we have Residuum rubrum (arterial and venous), and the extracts or powders formerly derived from rats and toads, or from parts and excretions of animals, are now carefully prepared and elegantly dispensed. Thus we now have—

Ox bile.	Pancreatic	Lymphatic
Pig bile.	substance.	gland substance.
Bone marrow.	Ovarian "	Mammary "
Cerebrin.	Spleen "	Pineal "
Didymin.	Spinal cord "	Pituitary "
	Uterine wall "	Prostate "

The search for specifics has always had a fascination, and the more intractable to treatment a disease seems to be, the greater the anxiety to obtain a remedy which shall be universally applicable. Such specifics are continually being advertised for consumption, but they rarely produce in the hands of others the success experienced by their discoverers. Perhaps the difference is to be sought in the general management or hygienic régime which accompanies the use of the remedy, —showing that the so-called “specific” is at the best only partially responsible for the success. We are all cognisant of the different effects produced by “taking the waters” at a well-organised “kurort,” and that which can be attained by taking the same waters at home.

In the last year or two the search for specifics has received a fresh impetus from our further knowledge of the action of micro-organisms. Antitoxins are being prepared for all diseases—tuberculosis as well as others. In discussing the use of these antitoxins it is necessary to bear in mind that in the treatment of poisoning more may be required than the administration of an antidote; important as it is to neutralise the poison, shock and local irritation, as well as toxic symptoms, may require attention. It may be that the antidote is itself a powerful poison, and danger may result from an overdose of the remedy. There is in these considerations no argument against the administration of the antidote; they merely emphasise the necessity for care in its administration, and suggest that we must not rely upon this alone. And further, as acute and chronic poisoning require different treatment, so also the success of antitoxins in some acute diseases does not necessarily imply similar success in tuberculosis.

The combined results of long-continued clinical observations in man, and of comparative observations in animals, show tuberculosis to be an infective process of long and persistent constitutional effects, and definite local lesions destructive of the tissues in which they occur. It is associated with and dependent upon a special micro-organism—the *Bacillus tuberculosis*. In dealing with the accumulated evidence from clinical observation and from experimental research the predominant interest must still attach itself to the disease as it shows itself among mankind. The relation between cause and environment is no simple question, and

each element needs separate study. The different behaviour of the same germ in various culture media, and the various effects produced in animals, are not without importance, but we must above all consider the differences observed in different human subjects under the influence of the same disease. Let us trace briefly the pathological changes which take place as a result of the invasion of the *Bacillus tuberculosis*—giving our attention primarily to the lung.

We will suppose the bacillus to have reached the bronchial tubes. Here, probably through some damaged epithelium, the bacilli get into the peri-bronchial lymph space, and multiplying set up inflammation in their immediate neighbourhood.

Cell aggregation and multiplication go on fast, and soon the mass of closely packed cells replaces all the original histological structure of the affected spot. Vessels are absent in the cell mass, and those in the immediate neighbourhood may become blocked and obliterated. The centre of the nodule is nothing but a mass of cells cut off from all blood supply, and forming a habitat for innumerable bacilli.

Caseation or destruction of the cells provides material on which the bacilli thrive, safe from the destructive action of living blood or of phagocytic leucocytes. The disease centre, impervious to both blood and lymph streams, is thus cut off from the influence of any antitoxin distributed by these fluids. This fact does not nullify the good effects of such remedies, but it certainly limits their operation. At the margin of the mass the bacilli continue the inflammatory action, and some of them getting into the blood or lymph vessels are carried away to start fresh foci of disease elsewhere. If now it were possible to keep the blood and lymph impregnated with antitoxin or antiseptic, such bacilli as get into these fluids might be destroyed, and extension of the disease be, to this extent, prevented. But even then the bacilli in the centre of the nodule still flourish, and are a continual source of danger. We cannot keep the blood charged with bactericidal substances ready to destroy the bacilli as they from time to time escape, for we should have to keep this up for weeks or months, with probable detriment to the body at large.

Thus, on theoretical grounds, we are led to

doubt the possibility of curing tuberculosis with antiseptics and antitoxins alone.

Whether it be possible to make the blood antiseptic by drugs at all may be doubted.

Again, let us suppose that the infective particles, whether introduced by the food or by the air, are arrested by the viscid mucus on the fauces. With undamaged epithelium the bacilli will probably remain innocuous on the mucous surface, and will be expelled or swallowed with the mucus. If, however, the epithelium be damaged—as by chronic pharyngitis, tonsillitis, or “relaxed throat”—the bacilli may find their way into the lymph spaces below, and pass into the lymph stream. Arrested at the first lymphatic gland they come into conflict with the lymph cells, and if not quickly destroyed by these cells may cause inflammation, perhaps leading to caseation of the gland (strumous gland). In some experimental work in which I was engaged a few years ago, I sprayed tuberculous material into the throat of certain rabbits, in some of which a subacute inflammatory condition of the fauces had been produced by repeated application of a weak iodine solution, others being untouched. The superficial cervical glands of the prepared animals, already somewhat damaged from the throat mischief, were found when the animals were killed to be enlarged, and tubercle bacilli were found in them. In the untouched animals these glands were found healthy. This demonstrates at least the possibility of strumous (tubercular) glands being caused by infection through the mouth. The caseation of one gland, accompanied as this change is with the multiplication of the bacilli, leads to the setting free of bacilli which may be carried by the lymph stream to the next gland, and there produce a similar condition of inflammation. But let us note a significant fact. Out of the many cases of strumous glands which we all meet with, how few, comparatively speaking, develop tubercular disease in other organs. True we often meet with cases of tubercular phthisis in persons of adult age—often of middle age—who in childhood have had strumous glands in the neck, evidenced by the scars which they carry through life. Are we to imagine that the bacilli have remained dormant for ten, twenty, or thirty years, and have after all this time become active, or may we not suppose a new infection from without? Seeing the ubiquity

of sources of infection, the latter supposition imposes less severe strain on the imagination than the former. In a patient recently under my care there was an interval of some twenty-five years at least between the strumous glands and the pulmonary tuberculosis.

The comparative rarity with which sufferers from tubercular disease of the cervical glands subsequently become phthisical has long forced itself on my notice. The significance of this has been explained by experimental research by which the power of living cells to destroy micro-organisms has been demonstrated. Here, then, is a most important fact. The healthy and vigorous cells in the body may and do safeguard the body from the effects of bacteria. Should these cells, however, be wanting in vigour, they are unable to prevent the development of bacteria which enter the tissues, and so to prevent disease. This is an elementary principle, and one which cannot be neglected in deciding upon the treatment of tuberculosis. If by antiseptics or antitoxins we could destroy all the bacilli in the body, we need take little notice of the vigour of the protective cells; for by the remedy we do the work which they should do, and it matters little then if they are unable to do their duty. But if the antitoxin can only reach a proportion of the bacilli in the body, leaving others out of reach which are actively engaged in their destructive work, we must beware that our remedy does not diminish the activity of the protective cells as well as of such bacteria as come under its influence. In an acute illness it may be possible to check the development and multiplication of the causative bacteria, and so cut short or cure the disease. If the protective power of the tissues is temporarily lessened by the treatment, as well as by the disease, invasion from without can be guarded against during convalescence. We have seen, however, that the struggle against the invading tubercle bacillus is necessarily prolonged; the invaders have a *piéd à terre* from whence they issue at intervals until all are destroyed or until their victory is complete.

We have clear clinical evidence that the tubercle bacillus becomes more potent for harm when the general health of the individual is lowered, or when a particular organ is temporarily or permanently damaged. We saw in the case of the rabbits in my experiments how the bacilli infected

those with damaged mucous membrane in the fauces, but not the others. In another series of experimental inoculations with tubercle, of rabbits in whom the lungs had been damaged, the control (or undamaged) animals which were inoculated became affected before the others. The damaged animals were strong and robust before they were operated upon; the control animals were poor ill-conditioned animals, though free from tuberculosis. This tends to show the greater susceptibility to infection of the weakly animals. Clinically we see the same thing in the cases of phthisis following influenza, measles, &c., or attacking those weakened by hysteria and depressing or exhausting work (*e. g.* long residence in hospital).

We might reasonably infer the protective power of healthy tissues from the effects of infection when resistance is diminished, even without the confirmatory evidence of Metschnikoff and others.

Reasoning from this it seems logical to endeavour to get the patient into good condition, and so aid his tissues to destroy the bacilli, rather than to trust to antiseptics or antitoxins, which may impair the protective power. This is, of course, difficult to accomplish when active tuberculosis keeps up a drain on the strength, but there is good hope of success in early stages. This line of treatment I have adopted with a satisfactory measure of success after some fifteen years' special experience, during which I have tried almost all the methods of treatment which have been advocated.

To lay down dogmatically the details of treatment is impossible, since, as we have seen, each case must be treated on its merits—the patient, not the disease. It is impossible to treat all patients alike. We cannot treat consumption, we must treat the consumptive individual. There are fashions in health resorts and fashions in drugs, but blindly to follow fashion is a confession of mediocrity. There is too much generalising in treatment where it is essential to particularise, but the latter requires more complete knowledge. Not only the seat, extent, stage and activity of the tubercular process require to be taken into account, but the temperament and peculiarities of the individual must be considered.

The principles of treatment are to build up the patient's strength, to relieve distressing symptoms, and to avoid infection of himself and of others.

To attain the first object a full supply of fresh air and good food with good hygienic surroundings are most important.

I would rather have to treat tuberculosis without drugs of any kind, than be restricted as to air, food, and other hygienic requirements, even with an unlimited command of drugs. The consumptive turned adrift far from civilisation in fresh pure dry air, and obliged to live the life of the woodsman and hunter, is better off as regards his chance of recovery than the miserable dweller in the heart of the richest city of the world, within reach of the most experienced specialists and of every drug, appliance, or "cure" which medical science (or anxiety) can suggest, but hampered by his unhygienic surroundings, by overcrowding, and by the necessary evils of the struggle for existence in a crowd.

Of all the various drugs which have from time to time been in vogue for consumption in the last fifteen years I have found little to choose between them in their effect on the disease. Some simple mixture, such as an acid with bitter, is often valuable to improve appetite and digestion, and is in my experience the most generally useful medicine in consumption.

Cod-liver oil and maltine are foods which are especially useful as additions to the ordinary dietary. The symptoms which often require to be treated are ineffectual cough, pain, diarrhoea, fever, and night sweats; most of which, however, generally soon improve under simple tonics, without special medication. For diarrhoea dieting and rest are important: for high temperature rest is essential.

The phlegm is a source of danger to the patient if it be swallowed, or if it is not cleared out of the air tubes. When expectorated it is a danger to others. It is to counteract these dangers that antiseptics find their chief value. By antiseptic drugs, inhalations, or vapours we may possibly do something to prevent auto-infection by sputa of intestine, lung, or larynx. By disinfection of the spittoon, the linen, and the room we may prevent infection of other persons.

We must, however, not deceive ourselves when speaking of the curative measures in tuberculosis. When it affects internal organs, such as the lungs, we are obliged to recognise that there is a stage in the disease beyond which we must not expect a

cure. Amelioration of symptoms, or quiescence of the disease process, may still be obtained for a longer or shorter period,—but with the ever-present risk of a recrudescence of the active mischief. For the successful treatment of pulmonary tuberculosis—and probably of all tubercular disease—early recognition of the existence and nature of the complaint is essential. The consumptive in the hands of a man who, with little knowledge of the art of prescribing, and small command of drugs, is able to recognise the presence of the disease in an early stage, is better off than one under the most famous expert, with the complaint in an advanced stage from want of early recognition. To treat successfully we must be able to recognise clearly. The stethoscope is so easy to use that no medical man is without one; the laryngoscope is less easy to manipulate, and is largely left to specialists.

Now I venture to say that, except for the initial practice in manipulation, it is far more difficult to judge of the evidence obtainable by the stethoscope than of that obtainable by the laryngoscope.

Still where we cannot cure we may prevent harm to others, and after all the highest aim of medicine should be to prevent disease. True, the longer a person suffers from tuberculosis the more risk there is of his infecting others, and thus an early cure has an influence in preventing the spread of the disease. But it is not by improved methods of treatment that the mortality from the most destructive diseases is materially lessened. Is it by improved methods of treatment that typhus has become so much rarer, or that ague has ceased to be a scourge in London and in the Fens? Neither is it due to any improvement in our treatment of tuberculosis that the death returns have shown a diminution of late years in the mortality from this disease. The greater attention to hygiene and sanitation is surely to be credited with much of the diminution of typhus, ague, and tuberculosis. Much more may be done to diminish the latter when we direct special prophylactic measures against this disease as has been done in the case of the other two diseases mentioned.

What these measures should be can hardly be laid down in their entirety by one individual, for they must include the management of animals as well as of human beings in their scope, and many interests will be affected and must be safeguarded.

Here personal experience cannot be invoked to endorse all the suggested measures. The success of prophylactic regulations cannot be decided by the experience of any one individual, and hardly by the united experience of a single generation. In deciding upon the measures to be recommended we have to study well the ætiology of the disease, and having learned the causes and mode of spread of the disease we base our regulations and instructions on these. Koch's discovery of the *Bacillus tuberculosis*, which for the first time demonstrated the infectious nature of the malady, showed the possibility of successful prophylaxis. Hitherto so little has been done in this country in the way of systematic prophylaxis for tuberculosis that we cannot do much more than theorise, though we have some valuable results in other countries (e.g. Germany) to guide us.

Experience will later on make it possible to improve—by elaboration or simplification—our original scheme. One thing of importance is to teach the public that tuberculosis is catching, and to get rid of immoderate fear of heredity.

Every physician may, however, draw from his case-book some cases bearing on the ætiology of the disease, and from these we may deduce suggestions which may save others from contracting the disease.

It is in the prevention of the disease that anti-septics will eventually find their true value in our fight against tuberculosis.

Glaucoma Treatment.—Whenever it is desirable to reduce ocular tension, a 1 per cent. solution of arecoline hydrobromide answers an excellent purpose. The instillation of a single drop gives rise to a burning sensation with lachrymation and blepharospasm; but these unpleasant results last no more than a minute; after them there are conjunctival hyperæmia with slight circumcorneal injection, but these, too, subside in a few minutes. At the end of two minutes clonic contractions of the iris occur, with diminution of the size of the pupil. The meiosis lasts for about half an hour at the utmost, and then the pupil gradually resumes its former size. The solution keeps well, retaining all its physiological properties for a year.—Lavagna ('Gaz. Hebdomadaire de Méd. et de Chir.,' May 1st, 1898).—*Monthly Cyclopædia.*

CHAPTERS FROM THE TEACHING OF DR. G. V. POORE.

No. V.

GENTLEMEN,—I now begin the more medical and technical part of the course. The medical jurist is more concerned with the dead than with the living, and therefore we have to consider the phenomenon of death. Life ceases not because we wear out, but because we break down. It may be that when public health reaches that pitch of perfection towards which some are striving, that we shall live indefinitely, and decay equally in every part, which will be a very doubtful gain. We live so long as the heart beats, and we have got to consider shortly why the heart stops. I will therefore review very shortly the main proximate causes of stoppage of the heart.

First there are mechanical causes, *a failure of the cardiac mechanism*. This may be *valvular*, as in sudden death in aortic regurgitation; you may get *blocking of the efferent vessels*, as in thrombosis of the pulmonary artery, to take a common cause; you may get air in the big veins so that the heart cannot drive the blood on; or, to take an exceptional case, you may get hæmorrhage into the pericardium and the heart stopped in that way. Or, again, the cardiac muscle may fail; it may fail from gradual degeneration, it may fail as a result of a poison, such as digitalis, which brings the heart to a standstill in systole, or aconite, which is said to bring the heart to a standstill in diastole. Then there are, of course, some organic poisons brewed by bacteria during infective diseases, such as diphtheria, which poison and stop the heart. In the same way you may, of course, get failure of innervation. The heart is said to stop sometimes from shock, and you sometimes hear—chiefly in the third volume of a sensational novel, I grant—of people dropping dead from mental shock. But you may take it that a person whose heart can be stopped in that way could not have lived much longer in any case.

We may get sudden death not from failure of the cardiac mechanism but from failure of blood, such as in sudden hæmorrhage or bursting of an aneurysm.

Then you may get failure of the pneumatic machine, such as paralysis of the diaphragm, from damage to the spinal cord, or from poisoning by such a thing as curare, which paralyses the end-organs in the muscles and so arrests their movement. Strychnia, on the other hand, causes tetanus of the diaphragm. Again, you may get a wound of both pleuræ, which makes the pneumatic machine useless. Or you may get disease of the lungs, those well-known pathological conditions, pulmonary apoplexy, double pleurisy, or double pneumonia. Then there are mechanical impediments to moving the chest. You may get exclusion of air from the lungs from a variety of causes—hanging, drowning, suffocation, substances impacted in the glottis, or membranous croup or diphtheria. There may be failure of oxygenation of the blood from damage to the hæmoglobin, such as in the inspiration of carbon monoxide. It enters into combination with the hæmoglobin, the aëration of the blood cannot go on, and so death results. There are cases on record in which failure of the pneumatic machine has been brought about by external compression, such as that produced by a plaster jacket. When a plaster jacket is put on, it is customary to put a towel underneath the jacket, which is ultimately withdrawn, so as to leave room for breathing while the jacket is on. There is a tale told of Horace Vernet, who had amongst the models in his studio a fine negro. He was beautifully formed, and they wanted to take a plaster cast of his chest; therefore they put on him a plaster jacket, and did not notice that he was getting dusky. They nearly killed him.

Again, we may have failure of the respiratory centre. This may be of cerebral origin. Or the respiratory centre is sometimes poisoned, and then it is said to be of hæmic origin. The centre is poisoned during the inhalation of chloroform, and it is poisoned in that condition which we speak of as uræmia. Of course the integrity of circulation and respiration depends really upon the health of the medulla oblongata, and it is said that you may slice the brain down to the level of the medulla oblongata, and you may slice the spinal cord away up to the level of the medulla oblongata, and that respiration and circulation will go on. The respiratory and circulatory centres are very near together, in the floor of the fourth ventricle, and sometimes one stops first, and sometimes the other.

All these facts which I have been recalling to your recollection are very necessary for you to remember as medical jurists when you come to make post-mortem examinations.

Bichat said that death comes on in three ways; that it approaches sometimes by way of the heart, sometimes by way of the lungs, sometimes by way of the brain. Well, when the heart stops first we get the phenomena which are common in syncope. The heart suddenly stops, and post mortem you find the body exactly in the condition in which it was at death—the cavities of the heart nearly empty, and the organs almost everywhere pale. Now when death occurs by the lungs you get a different set of phenomena, the phenomena of asphyxia. In death from the lungs the first feeling is one of want of breath and a sense of impending suffocation. Then you notice an ashy pallor, followed sometimes by a cyanotic blue tint in the lips and face. This gets darker and darker, and the respirations become gasping. Then the person falls insensible, and finally there are often convulsions. I should mention that in death from syncope there are often convulsions before death. It is important to remember that. Then, as I have said, Bichat described death as beginning at the brain. In death under those circumstances, one of the prominent symptoms is insensibility and very slow respiration. Well, death beginning at the brain is generally death from asphyxia; and in these cases of apoplexy you almost always find post-mortem evidence of asphyxia. It is convenient, for clinical purposes, to recognise these three forms of death—death beginning at the heart, at the lungs, at the brain; but for purposes of medical jurists, and for strictly scientific purposes, we must recognise that sometimes the circulation stops before the breathing, and that sometimes respiration stops before the circulation. When the action of the lungs has stopped, blood no longer passes through them, and as a consequence you get engorgement of the right side of the heart, and an engorgement of the veins throughout the body. But when the circulation stops before the respiration, then you get signs of pallor. It is very important to recognise these modes of the onset of death, and you must remember that in cases of failure of respiration the treatment is one thing, and in failure of the circulation the treatment is another. In a case of

syncope where the heart fails, the one thing which you have to do is to try to nourish the circulatory centre; and so you either put the patient on the floor, or depress the head between his knees, and hold him there until he begins to recover. The idea is to cause a determination of blood towards the medulla oblongata. Then give stimulants. Under this treatment, if he is recoverable, he comes round. Then in dealing with asphyxia, on the other hand, you must remember two things; to get the respiration re-established, so that artificial respiration is all-important, and if the right side of the heart and the venous system is unduly engorged with blood, because it is over-distended and cannot grapple with the work which it has to do, you can often do much in relieving the venous system by withdrawing a little blood. Never forget the importance of the respiration to the circulation; and that the lungs are (physiologically) inserted between the right and left heart. Respiratory movements can be artificially produced, and being thus produced will often start a circulation which is on the point of stopping. You will often do good, and can never do harm, by having recourse to artificial respiration in cases of lightning stroke, bullet wounds of the brain, &c. The experiments of Horsley and T. Oliver have shown this conclusively.

Now it is a very important thing in making post-mortems to know how death approached. I have here two rabbits, and the difference between them is that one has been drowned and the other has been suddenly killed by being knocked on the head. First of all we look at them externally. You will notice that the drowned rabbit has the eyes rather more prominent than those of the other rabbit. Sometimes you may notice a difference in the position of the tongue—sometimes the drowned animal has the tongue thrust forward between the teeth. In these animals, however, there is but little difference in that respect. I now proceed to open them. I ask you to notice the difference in the tint of the intestines. In the drowned rabbit the intestine is of a dark dun colour, whereas in the other animal it is of a comparatively light grey; the difference is most marked in the small intestine. Looking at the drowned rabbit you are justified in saying that death was the result of failure of respiration, judging by the colour of the viscera. If you say, as I have no

doubt has been said hundreds of times, "Oh, these engorged intestines show inflammation of the intestine, possibly from poisoning or something of that kind," that is a conclusion which is quite unwarrantable and wrong. We next come to the heart. Here, again, in the rabbit which was suddenly killed the right ventricle is of about the same size as the left, but in the drowned rabbit you will see the right ventricle is much more distended than the right ventricle of the other animal. The lungs in the killed rabbit are pale, while in the drowned one they are somewhat engorged. But you must remember that the condition of the lungs in death from asphyxia is variable, and the condition of the left ventricle in asphyxia is also variable. It is said that if you make a post-mortem immediately after drowning, you often find the left ventricle distended. But as rigor mortis comes on the left ventricle empties itself and the right ventricle is engorged. Here the right ventricle is obviously engorged. The point is this. You assume these are two people found dead, and you are asked by the coroner to make a post-mortem examination. You open the body and find a general pallor or general engorgement of the viscera, as the case may be. If you find general engorgement of the viscera, you search for whatever may have caused this engorgement, and that may have brought the lungs to a stand-still. You may find evidence of drowning, you may find a foreign body in the trachea, or you may find, as I did once, death from membranous croup which was unsuspected. Remember that the causes of this engorgement and consequent failure of respiration may be found in the kidneys. In "uræmia" you may get the respiratory centre poisoned and a gradual failure of respiration. In opium poisoning you get the same thing, respirations getting slower and slower and an engorgement of the veins throughout the body. Sometimes it is stated in works on medical jurisprudence that opium causes engorgement of the vessels of the brain, but it only causes that because it produces slowness of respiration and brings about death from gradually supervening asphyxia.

I would allude once more to the fact that in death occurring by the heart, the lungs, or the brain, convulsions may equally occur. Because a person is comatose or because he is convulsed, do

not rush to the conclusion that therefore the cause of death is in the brain. That is a conclusion that is not at all warrantable. In no disease is coma more marked than in diabetes, but that is a disease of the blood or of the viscera, and not of the brain. Again, in uræmia you may open the brain and find it quite healthy. On the other hand, you may get a severe cerebral hæmorrhage leading to pronounced hemiplegia, and yet you often find that the patient suffering in that way is not comatose, is not delirious, is perfectly conscious, and not convulsed. Therefore do not necessarily attribute coma and convulsions to primary brain disease. Of course you would not have the coma or the convulsions if the brain were properly nourished, but do not attribute such things to *primary* brain disease.

Now what are the signs of death? I do not think they need detain us very long. When a man is dead the circulation ceases and the respiration ceases, and it is very seldom indeed that you have any difficulty in saying whether a person is dead or not. Occasionally it happens with very old people that death advances very very slowly indeed, the respirations get slower and shallower, and finally cease, and in watching a person in this way you cannot quite say when death took place. And in cases of this kind sometimes friends are nervous and think that death has not taken place when really it has. If deaths of this kind take place in subjects not prone to putrefaction, and at a time of year such as the depth of winter, when putrefaction is held in abeyance, it is difficult to convince people that their relative is really dead. There are cases on record in which people have remained with scarcely perceptible movement for a long time and have recovered, and we have in the hibernation of animals a condition in which respiration gets excessively slow, and the condition really approaches that of death.

When you are called to a case to know whether a person is dead or not, can you determine it? Of course you listen to the heart, you watch the respirations, you feel the pulse. Then, if still in doubt, a good plan is said to be to tie a ligature round the root of the finger, and if the circulation is going on the strangled finger will get blue. There is a source of fallacy there. I remember once on visiting the hospital one of the house physicians came to me and asked me to go

upstairs. He said there was a man just dead, and he had tied a ligature round his finger and it had got blue, nevertheless certainly the man was dead. I cross-examined this gentleman and found that he had not merely tied the ligature round the finger, but had done everything he could to draw blood into the extremity by a process akin to that employed in milking a cow, and by that means he had made a blue finger, but the circulation did not make it. If that test is to be of any use you must merely encircle the finger with a ligature and nothing else. Sometimes people think that there has been vital movement of the body after death, and it seems very generally admitted that after sudden death from some diseases, especially cholera, there are movements of the limbs.

Another test is said to be to apply a blister, when if a person is alive the blister will rise, and if the person is dead the blister will not rise. Another test of use is the loss of muscular irritability. Immediately after death the irritability of the muscles remains, and with a faradising machine you may get a little contraction; but after death has taken place some hours there is no longer any contraction of the muscles.

Now there are certain conditions of the dead body which enable you to form a judgment as to when death took place—how long a body has been dead. One circumstance is the cooling of the body. Some of the books have said that the body cools at the rate of 1° F. per hour. That is so easy to remember that it is a pity it is not true. But you have only to think of it to see that it could not be true. The rate of the cooling of the body depends upon circumstances, and mainly on the difference that exists at the time of death between the temperature of the body and the temperature of the surrounding air. If a febrile body is left exposed to the extreme cold of a winter night and be naked, cooling will go on exceedingly rapidly; if the body be a fat one, and be beneath two or three blankets and an eider-down quilt in a hot room, cooling is naturally very slow, so that you cannot lay down any rule of any kind whatever.

You must therefore take the whole of the circumstances into account, and I would warn you when you go into courts of law not to attempt to be precise where precision is really impossible. If you try to lay down rules of that kind you will, with

the best intentions, more often deceive the jury and yourselves than you will do good.

Now I would remind you that sometimes after death the temperature rises. Of course that is a very complicating circumstance. At the moment of death the temperature may be 106° or 107° , and after death it may reach 112° or 114° , as has happened.

The internal warmth of the body is very often perceptible to the hand twenty-four hours after death; indeed, in making a post-mortem examination twenty-four hours after death, you may find the viscera perceptibly warm.

After death there is another phenomenon which we must attend to, namely, rigor mortis. In rigor mortis the muscle becomes shorter, harder, and inelastic; it becomes lightly acid in reaction, and opaque instead of translucent. Rigor mortis is due to the coagulation of muscle plasma. As a rule the muscles remain in the position in which they were at death. But that is not quite true. You will find, for instance, that there is what is known as the cadaveric position of the hand (slight flexion of the thumb and fingers), due to the contraction of the muscles after death before the extinction of muscular irritability, in which the muscles are subject to the action of the spinal cord only, and the stronger group get the mastery. You will find that this position is assumed against the force of gravity. The time of onset of the rigor mortis varies, but as a rule it is said to begin five to six hours post mortem. But this, again, is so variable that there can be no rule at all. It would appear practically that the time of onset of rigor mortis bears a direct ratio to the irritability of the muscle at the time of death. If the muscle be in good condition with its normal irritability, rigor mortis sets in slowly; if the muscle is exhausted, rigor mortis sets in early. Now in persons who die while undergoing great muscular exertion, the muscles are sometimes fixed at the moment of death apparently. Whether this be precisely analogous to rigor mortis, or whether it be a spasm occurring at death which continues until that spasm is fixed in rigor mortis, is a debated point. As a practical fact, when the muscles are exhausted by excessive work at the moment of death, rigor mortis or cadaveric spasm comes on instantly. The hare which has been coursed to death stiffens very quickly because the muscles

are exhausted, and rigor mortis sets in instantly and passes off quickly. The coursed hare may therefore be eaten by the epicure earlier than a shot hare because it is fit to cook earlier. So also on the field of battle after forced marches, or when a man is killed while fighting for dear life and putting forth a quite unusual amount of muscular energy, it is found that he remains fixed in the position in which he was when he was killed. That is apparently due to the exertion, and possibly also to the way in which he is killed. A man shot through the head is very often fixed in the position in which he fell, and I take it that the evidence is perfectly good that soldiers have been found grasping the rifle and in the position adopted for firing, perhaps in readiness to shoot over an earthwork. There is a case which comes from Germany of a man who got out of a train at a railway junction and went across the metals to the refreshment room; he came out of the refreshment room with a sausage in one hand and a sandwich in the other, and on going back to his carriage his head came between the buffers of opposing carriages and he was killed instantly. When an examination was made the sausage and the sandwich were tightly grasped in the hands. That was due to some spasm of the muscles caused by the shock to the brain. So it happens sometimes in death from lightning that rigor mortis comes on suddenly. The sooner rigor mortis comes on the sooner it passes off; that is the rule. John Hunter came to the conclusion that in death from lightning rigor mortis did not set in. The reason for that belief was probably that it set in early and passed off early, and at the time of inspection it had ceased. And so when patients die of exhausting diseases, with presumably little muscular irritability left, the rigor mortis sets in early and passes off early. That is one of the difficulties with people who die gradually. In those cases rigor mortis either does not come on or it is badly developed and passes off quickly, and people think that the body is not dead because it has not got stiff.

Now there are certain relationships, perhaps more apparent than real, between vital contraction of a muscle and rigor mortis. I will remind you that after excessive exercises—we all must have experienced it after a walk of unwonted length—when after a rest we rise to continue our journey,

our walking is labour and sorrow, because the muscles are all stiff. That is a matter of common occurrence, but after walking a little way again the muscles become supple. Now after exercises a muscle is acid in reaction; a muscle in rigor mortis is acid in reaction. A muscle after excessive exercises is stiff, it does not move easily, it requires force—sometimes considerable force—to move it; the muscle in rigor mortis is stiff in the same way. Now whether these be the same or allied conditions or not, it seems perfectly certain that the muscle which has been excessively used immediately before death passes quickly into rigor mortis, the best example being the coursed hare, and the next example being the death spasm as seen in the dead soldier shot through the head; but it is doubtful whether the physiological condition of the coursed hare, which is a matter of fatigue, and the physiological condition of the dead soldier is the same thing. You know that when we are very intent upon anything we put forward an enormous amount of muscular power, very much more than is necessary. This probably occurs in battle. When a muscle is severed from the nerve centres, rigor mortis is late in coming on. That is what might be expected, because a muscle separated from the nerve centres cannot get fatigued, and cannot get spasm, and cannot be subjected to those conditions which practically we know precede the early onset of rigor mortis.

The next point is, why does rigor mortis go off? Some say it goes off because of the onset of putrefaction. That is open to question, but it is nevertheless true that after rigor mortis has gone off putrefaction usually sets in early. The coursed hare must be eaten the day it is killed, or within twenty-four hours; if it is not it goes putrid; that is a well-known fact. I take it that the difference between tender and tough meat is whether you eat the meat before the rigor mortis has gone off, or whether you wait until the rigor has gone off and the muscles become soft again. Rigor mortis once destroyed does not return; therefore the cook takes a steak and beats it, and she makes the steak tender. Now cadaveric spasm may be of importance in law courts. There is a case recorded from Bordeaux which is of importance in this connection. A man was found by his bedside sitting in a chair, dead, with a bullet wound through his head. The

dead body was leaning against the bed, and across the right knee was his right hand, and in the hand a pistol. Of course the case looked like one of suicide. But the son who slept in the same room was a *mauvais sujet*, and question arose whether he had not murdered his father and put the pistol in his father's right hand in order to deceive. The first person who came into the room jogged against the corpse and the pistol dropped from the hand very readily, and the arguments *pro* and *con.* turned upon that. The pistol was very loosely held. Could it have been put there by the son? Or was it a true case of death spasm holding the pistol? Now if you should be called to a dead body in which a similar condition of things exists, you must examine it very carefully for cadaveric spasm. The question is, can it be imitated. I should very much doubt whether cadaveric spasm can be imitated, and I should think there are cogent reasons against its possibility. You may put the dead hand over a weapon, and pull the fingers over the weapon and leave it. By doing that you have fixed the finger-joints, but you have done nothing to the flexor muscles of the fingers. The flexors of the hand not being in contraction would not be kept in contraction. I should say it was impossible to imitate a death spasm by a trick such as I have suggested. Always examine to see if the muscles are really stiffened.

Stuttering.—Liebmann maintains that stuttering consists in a rushing together of the consonants, and consequently his method of treatment has for its object the teaching of the relative significance of the vowel and consonant sounds. The patient is made to speak sentences with prolonged vowels and short consonants, so that even at the first lesson many sentences are spoken easily and fluently. The psychic effect is soon apparent, the patient regaining confidence in his ability to speak plainly, and the result is excellent in a very short time. This method can also be employed with young children. Stuttering may be caused by the infectious diseases, injury to the head, imitation of other children, or by heredity. Its more frequent occurrence in males than in females is to be explained by the greater motility of all the voluntary muscles in women than in men, the tongue included.—*Der Kinder-Arzt.*

FEMORAL HERNIA IN MALES.

A Clinical Lecture delivered in the Post-Graduate Course at the West London Hospital, Hammersmith, on Wednesday, June 15th, 1898, by

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GENTLEMEN,—I have this afternoon to show you a number of cases illustrating the subject of femoral hernia as it occurs in males. There is no doubt that femoral hernia is more often found in the opposite sex, but even in females, if we take all ages into consideration, femoral herniæ are not so common as inguinal. Of every 100 instances of females afflicted with hernia, 60 have inguinal hernia, and therefore only 40 have femoral. This, I think, you will see is not in accord with the ordinary teaching. But when we come to look at the other sex we find that out of 100 cases of hernia in males 97½ per cent. are of the inguinal type, while only 2½ per cent. are femoral. This shows a very great difference between the sexes. Still, seeing that there are many more males than females affected with hernia, it follows that male subjects form a not inconsiderable proportion of the persons who are labouring under femoral herniæ. I will not delay to enter upon the causes of femoral hernia in males, but I will, with your leave, bring before you some actual cases which will of themselves illustrate the points which I desire to emphasise.

The first patient presents a typical small right femoral hernia. I ask you to notice the position of the hernia, lying altogether below Poupart's ligament. You will observe its characteristic form, a rounded swelling, not oval nor pyriform. It is quite easily reducible, but it readily descends again on coughing. It is, however, restrained satisfactorily by an ordinary right 32-inch femoral truss. Here I draw your attention to the proper shape of such an instrument, and also the right way in which it is to be measured for. You will see what I mean by a sketch on the board. I would particularly ask you to notice the line of the stitching and the position of the studs. I pass round an ordinary femoral truss and a photograph of a femoral and an inguinal truss side by side. In the femoral truss you will see that the pad is smaller and the lower part has

a very distinct curve, whereas in the inguinal form the pad is larger and the curve is very slight. This proper shape, therefore, is not that which is often depicted in the books. You will see from this drawing that the difference between the position of the inguinal canal and the femoral ring is only the difference of the thickness of Poupart's ligament. Therefore if we put on an inguinal pad in its correct position it will lie over the deep or internal ring and canal, and the femoral pad would lie not much lower down, but more internally. As to the measurement of these cases, you proceed in exactly the same way as for an inguinal truss, that is to say, starting behind at the base of the sacrum, and coming obliquely round the pelvis, your measure being parallel to its plane, and meeting in front above the level of symphysis pubis. I think the best way is to place the small numbers of the measure nearest to the patient's skin, holding one end over the deep abdominal ring, bring the other end to meet it by putting your thumbs together, and then drawing the measure away, your right thumb marks the spot which indicates the measure. This man measures 32½; we cannot call it 32, for then the truss would be too tight, therefore we call it 33. This is a typical case of femoral hernia in its early stages; just a slight fulness, but if you put your finger over the ring and get the patient to cough there is an expansile impulse. Such a small hernia is particularly liable to be overlooked in male subjects. Femoral hernia in a man is very dangerous; it is prone to become strangulated and otherwise cause trouble. A truss must always be adjusted carefully with the patient recumbent, the hernia being reduced as far as possible before the truss is applied. It is applied obliquely, and the under-strap is brought from the shoulder of the truss and over the perinæum, up to the upper button of the pad.

The next is also an interesting case, showing left femoral hernia, reducible, but in a more advanced condition. Here you see a swelling on the upper part of the thigh, rather to the inner side, distinctly below Poupart's ligament, not mounting up on to the abdomen, as femoral herniæ are said so commonly to do, but remaining as a globular swelling in the thigh. On coughing you will notice that the tumour is distinctly expansile. When a male suffers from femoral hernia it not infrequently transpires that one of the females of the

family were or are ruptured. I simply bring this case forward as a more advanced form of the affection. In this patient, after reducing the hernia you can feel the sharp edge of the saphenous opening. If you gently put your finger up through that aperture you will feel the femoral ring itself. It is important to remember that a truss must be worn next to the skin; if it is worn outside any article of clothing every movement of the body and therefore of the clothing causes displacement of the pad. There is a little tendency in this case for the hernia to bulge below the pad. It is an advancing femoral, and will probably shortly require another form of femoral truss.

I will now pass to the consideration of some forms of irreducible femoral hernia. Here is another patient showing the usual form of small femoral swelling, hardly perceptible at a distance; but in this case instead of being reducible, as in the first two cases, it is irreducible. You will find that very frequently femorals become irreducible, and their irreducibility is most often due to adherent omentum. Not infrequently you get a development of fluid in the sac below the adherent omentum, so as to produce a kind of hydrocele of the femoral hernial sac.

For the treatment of irreducible femoral hernia of small size we use what is known as the hollow pad truss, a specimen of which I show you here. It is like an ordinary femoral truss, but the pad instead of being convex is hollowed out. That exercises a great deal of continuous pressure on the hernia, and in the majority of cases reduces it.

As the hernia goes gradually back one fills up the hollow with a little cotton wool, procuring more and more pressure until at last it all goes back, and then you can put on an ordinary femoral truss. With an irreducible femoral you will generally get an expansile impulse when the patient coughs, because although what is in the sac cannot get back, yet very frequently more can come down. If it is completely shut off from the abdomen by adhesion below you probably get no impulse at all.

The next case is a man who has a double femoral, by which you will see that men have double femoral hernia as well as women. You see a bulging in either femoral region below the line of Poupart's ligament. He also has a funnel-shaped projection running down to the external superficial

abdominal ring, which is not at all uncommon in the subjects of hernia. You will notice he is wearing a double femoral truss of the approved type.

The next patient came up to the Truss Society the other day with a hernia which at first looked exactly like a double femoral; but if you carefully examine the patient you will see he has a very oblique pelvis, and that it is really an inguinal case. I will reduce the hernia, and if I now place my finger over the canal and the rings the protrusions do not come down. When I first saw the case there was nothing down in the scrotum. He is wearing a double-forked tongue truss, the whole of the tail portion being made of soft material, an important point often neglected in the construction of such trusses.

The next man is the subject of a femoral hernia somewhat larger than the ones I have been showing you. I will ask you to look at his truss before I take it off. It is spoken of as a femoral, with this portion which is called a thigh belt, and the part I show you here is called the inguinal fulness. If you have a femoral hernia which tends to mount up in front of Poupart's ligament and therefore to obscure the ligament, you must add an inguinal fulness to your truss. On the other hand, if you have a femoral which tends to go down the thigh, you have to order a thigh belt. An inguinal fulness ought never to be put on without a thigh belt, but you may have a thigh belt without an inguinal fulness. You measure in exactly the same way as for an ordinary femoral, taking the measurement round the upper part of the thigh for the thigh belt. The thigh belt is attached to the piece of soft material which projects below the ordinary pad. He was wearing an ordinary femoral truss which failed to keep up this hernia, which has a great tendency to mount up in front of Poupart's ligament and to pass down the thigh. Quite apart from his hernia, this patient is the subject of a very interesting condition on the opposite side, a condition which I confess I have only once before seen. As far as I can make out it is a calcification of the wall of an old hæmatocele. It has been suggested that it might be an enchondroma of the testis, which is an extremely rare condition. I do not think it is that, because it does not involve the whole body of the testis and the epididymis, as an enchondroma usually does. You will feel that it

is a very hard swelling indeed. The history is that at the age of thirteen he was struck on the right testis by a cricket ball, and shortly after that, that is to say within a few hours, a very large swelling occurred, attended by extreme pain. The swelling never completely disappeared, but the pain went, leaving the hard tumour. I intend some time to throw it on the screen by the Röntgen rays. If it is calcification it will throw a distinct shadow, but very little if it is cartilage.

I have now dealt with the subject of femoral hernia existing alone, both single and reducible and irreducible, and then double. I will now show you some instances of femoral hernia combined with inguinal. This man has a double femoral hernia with a right inguinal. In most of these cases of combination of femoral with inguinal the femoral herniæ are the ones which give rise to the greater trouble. One, therefore, sometimes disregards small inguinal herniæ, and deals with the more important femoral protrusions. This patient is wearing a double femoral truss. On the left side the femoral is irreducible, and he is wearing a hollow pad. The use of the under-strap is to pull the truss away from the crest of the ilium, and at the same time to give the pad a tilt upwards, because when we are standing the femoral ring is almost horizontal.

The next patient has a right scrotal hernia and a left femoral. When a man has a scrotal hernia and a femoral which requires a thigh belt, it means that he has been neglecting his herniæ, or perhaps has not had the opportunity of having a proper kind of truss. Therefore one has to put on in this case what appears to be a very cumbersome form of truss, viz. a femoral pad with a thigh belt and an inguinal fulness over on the left side with a forked tongue on the right, in order to get the herniæ into a better condition. Many of these herniæ when not properly treated in their early stages become large and almost unmanageable. I show you how to fasten it, and pass round photographs of such trusses.

The next man has a double inguinal and a right femoral. As I have said, one often disregards the inguinal in these cases and adjusts a double femoral truss, raising it on the side where the inguinal is most marked. His truss is now in such a position that it controls the inguinal. The cross-strap should come from the lower stud, so that it

raises the pad a little on the inguinal side. Strictly speaking, this is not altogether scientific, but the distance between the inguinal opening and the femoral opening is so slight that in a great many cases one can safely use a double femoral truss for an inguinal on one side, and a femoral on the other. However, perhaps it is better to have a truss made with the pad on one side inguinal and the pad on the other side femoral. This man also has a small umbilical hernia. It is interesting because he is now forty-three years of age, and he has apparently had the umbilical hernia ever since infancy. It is not common to find umbilical hernia of the acquired infantile form—often, but wrongly, called “congenital”—persisting throughout life. Every patient who comes to the Truss Society with an umbilical hernia is asked how long he has had it, and this is only the third or fourth case in which I have got a distinct history that it has been present from infancy. The reason is that almost always umbilical hernia becomes cured. I have given him a small umbilical truss.

I will now show you some rather out-of-the-way forms of femoral hernia. Before I take the truss off I ask you to notice it. It is a double femoral with a double inguinal fulness over, and a double thigh belt, obviously meaning that he has considerable protrusions. This man was operated upon thirteen years ago, and I need hardly say that one gets all sorts of fantastic shapes of herniæ after operation. You will agree that it is a very large hernia and extremely pronounced. It looks almost as if it were an inguinal hernia, but you will see it lies practically altogether below Poupart's ligament. The scrotum you see is entirely unaffected. When he lies down and coughs you see a typical femoral bulging. You can feel the femoral ring, and you can readily feel Gimbernat's ligament and realise how it would cut into the gut if strangulation occurred. With regard to the other side, you can feel exactly the same position of affairs. Below is a huge opening through which I can easily get three fingers. That is one of the results of “cutting the stricture,” as it is called, in a case of femoral hernia. My own feeling is that one should as far as possible avoid cutting any stricture in strangulated herniæ. Dilate the parts as much as you like by stretching them, and they will afterwards retract. By cutting them you are liable to leave a large opening, unless of course you proceed

to a thorough radical operation, which one cannot always do. The reason this hernia is so big is because of the enormous opening, and because by pressure has been developed what resembles a second scrotum, into which the hernial sac protrudes. It not only comes down into that sac, but it passes up towards the anterior superior spine, so that the hernia almost reaches that point of the ilium. I have seen many femorals in which no operation has been performed pass down the thigh; in fact, femorals do not always ascend in front of Poupart's ligament, as one would rather expect from statements made concerning such protrusions.

The last patient I have to show you is one who came to the Truss Society yesterday morning. I will ask you to first of all notice the truss he is now wearing. I think it is supposed to be an inguinal truss—that is to say, a rat-tailed inguinal truss. In scrotal herniæ the metal portion of the pad must never be prolonged into the tail portion, because it would press the spermatic cord against the bone and produce agony to the patient. If you will examine the patient closely you will agree with me that his hernia is a femoral one on the left side. On the right, however, he has an inguinal protrusion. Here, again, the femoral hernia does not come up over Poupart's ligament, but is tending to drop down in the thigh. He has been an old friend of ours at the Truss Society, but last time he did not come back to us for a truss, and he does not appear to have obtained one which corresponded to his hernia. In measuring for a double truss remember to add one inch to the measurement, because such a truss does not stretch as will a single one.

I have not referred to the question of radical operation for femoral hernia because it is too large a subject to deal with this afternoon, and therefore I have not brought before you any case in which the operation has been done.

[A large number of trusses illustrating the different forms suitable for femoral herniæ were then demonstrated.]

NOTES, &c.

Acute Chloral Dementia simulating Paretic Dementia.—Drugs play an important rôle in the pathogenesis of mental states, and the clinical picture presented by these varying conditions is often puzzling and difficult to analyse. Chloral is frequently employed by drug *habitués*, and its symptoms are often misinterpreted with consequent erroneous diagnosis and prognosis. These often involve the rights of the patient.

The basic principles of paretic dementia are well known, and are in the initial stages more or less pronounced ataxia, delusions of a grandiose type, fibrillary or general tremor, hyperhydrosis, and pupillary inequality. Evidences of dementia are found which later become pronounced. Not infrequently insomnia is a prominent symptom early in the affection. A patient coming under observation suffering from such symptoms is naturally classed as a paretic. In some of these cases it is found that chloral has been used for obtaining sleep for weeks before the patient comes under observation. The prompt withdrawal of the drug is often followed by a disappearance of the symptoms and the restoration of the patient to mental health. The asylum records of this country show a considerable number of incorrectly diagnosticated cases of acute chloral dementia in which a short residence in the asylum has resulted in a cure, and the patients were discharged with the stigma attached to them of having been committed to an insane asylum, an error that might have been avoided by a correct appreciation of the ætiology of these symptoms.

It would seem wise to keep cases in which hypnotics have been employed under observation a sufficient length of time to learn if perchance it may not be a case of drug-habit insanity. The fact that even learned alienists may be mistaken in the diagnosis of these cases emphasises the necessity of the ordinary practitioner exercising great caution in their commitment.

Medicine, August, 1898.

Suppository Doses for Children.—In the May number of 'Pediatrics' is an article, abstracted from 'Médecine moderne,' calling attention to the many advantages of rectal medication in children, viz. lesser irritability of the rectum than that of the delicate digestive organs, emotional antagonism of the child, &c. Rectal absorption is perfect, but slower than that of stomach and intestines, whence the dose may be as large as that given by the mouth, and gradually increased. If the drug is dissolved in the suppository its effect is attained gradually. The following are recommended as

the maximum doses of certain potent remedies for children:

Opium.—Pulvis opii may be given in suppository in doses of one sixty-sixth of a grain for each year of the child's age, and this dose may be repeated in severe cases every two hours. Toxic symptoms should be carefully watched for, and the use of the remedy discontinued on their appearance. These doses are small ones, and may be increased.

Aconite shows its action in children only in large doses. We must therefore administer it in repeated small doses to obtain its effect. For example, we may give one or two drops of the tincture for a suppository in a year-old child, and increase the dose up to ten or twelve drops in twenty-four hours for each year of life.

Belladonna acts as an excellent sedative in cough, and exerts a very favourable influence on the muscle-fibres of the intestine. We may use one sixth of a minim of extract of belladonna in twenty-four hours, divided into three or four suppositories, for every two years of age.

Digitalis.—Powdered digitalis is with difficulty absorbed by the rectum. The tincture should therefore be used. The maximum dose for each year of life is four drops, divided into two suppositories.

Caffeine is usually injected subcutaneously. It may, however, be administered in a suppository with equal parts of benzoate of sodium; for example, one grain and a half to a suppository, using two daily for each year of the child's life.

Quinine is best given in suppositories. The daily maximum dose is two to three grains and a third, in two suppositories, for each year of life.

Antipyrin may be given in the same dose.

Salicylic acid.—Seven grains and three-quarters for each year of life, in divided doses (three or four).

Nux vomica.—One sixth of a grain for every two years, in three suppositories.

Strychnine should be given only to children over ten years of age.

Mercury should only exceptionally be given *per rectum*, and then only in the form of calomel, three quarters of a grain in a suppository, for each year of life.

Iodine and its preparations are exceptionally well borne by the rectum and fully absorbed. Three grains for each year of life, in two suppositories, is the maximum dose; three-quarters of a grain if it is to be continued.

Bromides should be given in the same doses, except in severe spasms, when we may give fifteen grains, for each year of life, in two suppositories rapidly following each other; for instance, in laryngismus stridulus.

University Medical Magazine.

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ON PRACTICAL POINTS IN PERCUSSION AND AUSCULTATION, AND IN THE PHYSICAL EXAMINATION OF THE CHEST.

ABSTRACT OF LECTURES TO ADVANCED CLINICAL STUDENTS

BY

WILLIAM EWART, M.D., F.R.C.P.,
Physician to and Joint Lecturer in Medicine at St. George's Hospital; Physician to the Belgrave Hospital for Children.

IV.

ON PULMONARY AUSCULTATION, WITH REMARKS ON MEDIASTINAL FRICTION, ON AUSCULTATORY PERCUSSION, AND ON AUSCULTATORY FRICTION.

The Anatomical Basis of Pulmonary Auscultation.

THE solid conduction of the skeletal structures for vibrations is in connection with auscultation both a help and a possible source of fallacy. We shall find it frequently complicating the results of our auscultation; but the influence of pleximetric conduction is more obvious in connection with the method of auscultatory percussion which we shall presently consider.

A good auscultator should realise vividly the underlying material; he should be familiar with the boundaries of the lung, with the varying thickness of its edges, and with the position of its lobes.

We have already taken note of the small size of the apex of the lung. Bear in mind that the left apex forms a smaller and more acute pyramid, the right a broader one, in connection with the left-sided position of the great vessels—the aorta, the pulmonary artery, and the left jugular. The lowermost limit of the base of the lung is posteriorly the twelfth rib. Between that level and the level of the infra-sternal notch, the pulmonary boundary follows an oblique line which will variously intersect the several ribs according as they slant

more or less in the individual. The lower edge is thin and fits into the diaphragmatic groove, but the arching under-surface represents an enormous reserve of breathing available for extraordinary exertion, and is also utilised by singers taught according to the modern school to "breathe from the stomach." This implies, of course, some lower costal breathing, and therefore necessitates the avoidance of any constrictive pressure at the waist.

Passing now to a consideration of *the lobes*, we find that *anteriorly* the lower termination of the main interlobar septum is nearly symmetrical in the two lungs, occupying at the base a spot slightly external to the vertical nipple line. Between this and the sternum, the heart and the cardiac lobule of the upper lobe intervene on the left side, and the middle lobe on the right.

On the right side the short horizontal interlobar septum which forms the upper boundary of the middle lobe extends from the posterior axillary line along the fourth rib and cartilage to the sternal border.

A knowledge of these relations has its practical importance. Thus in pneumonia of the base of the right lower lobe you will find the physical signs limited in front by a line passing from the foot of the vertical nipple line backwards and upwards to the point where the fourth rib crosses the posterior axillary line, which is also the point of junction of the horizontal septum and of the main interlobar septum.

Posteriorly on both sides there are only two lobes to be seen, but they are not quite symmetrical. The right interlobar septum begins at the level of the fourth spinous process with a gentle curve which slopes downwards with less declivity than on the left side. The left interlobar septum is more convex upwards, and rises to the level of the third spinous process; it then falls with greater steepness, so that the left upper lobe viewed from the back has a greater vertical length than the right, although its apex is shorter. Correspondingly the left lower lobe has a considerable length from its apex to its base, whilst the right lower lobe is not quite so long.

As pointed out to you previously, on either side of the spinous process nothing is to be seen of the underlying structures but lung only, under cover of the ribs.

The Local Differences in the Respiratory Sounds.

The functional respiratory value of the various parts of the chest, and the characters of the respiratory sounds are not uniform. Unless we know what we may expect to hear at each part of the normal chest our auscultation might be misleading.

The differences in the sounds are largely due to the local differences in the respiratory mobility of the thorax. Thus the apex proper has but a small respiratory excursion; the pectoral region is an active respiratory district, particularly in the female; and the dorsal region has considerable respiratory capacities usually kept in reserve. The lateral regions are exceedingly active, and respond almost the earliest to any special call for breath. The basic regions are capable of very great activity, and in the male the diaphragm regulates the ordinary supply of tidal air, though it frequently alternates this office with the pectoral region.

The "Special" Auscultatory Areas.

As in percussion, we have to take note of the peculiarities of certain specialised regions of which the following is a list:

1. The supra-clavicular and the infra-clavicular,
2. The manubrial and the upper parasternal,
3. The supra-spinous,
4. The vertebral (both cervical and dorsal), and
5. The interscapular areas.

Our acquaintance with the pleximetric conditions which influence percussion will stand us in good stead; for, thanks to the stethoscope, which collects and transmits them, the auditory vibrations seem to be conducted along the solid parts of the chest even more perfectly than those elicited by ordinary percussion. Bearing this in mind, our knowledge of the relations of the lungs to the bony thorax and to the neighbouring organs will help you to understand, nay almost to predict, what auscultatory peculiarities are special to each district.

The supra-clavicular region, for instance, is distinguished by its powerful conduction for the bronchial or tracheal elements of breathing, whilst the vesicular murmur is not much to the fore, seeing how little inspiratory expansion is effected by the practically fixed first rib. The respiratory

type of the supra-clavicular fossa would thus be the broncho-vesicular type, which in many individuals is much more bronchial than vesicular, and in some is loudly tracheal. Beware of taxing subjects of this kind, who are probably perfectly healthy, with being the bearers of the bacillus.

What applies to the breath-sounds applies also to the vocal resonance. A spurious whispering pectoriloquy may even be traced in some thin people in this situation.

In the infra-clavicular region, including the first rib and the first interspace, the respiratory murmur often partakes of this conducted bronchial character. A full tracheal respiratory sound is not, however, commonly to be heard.

Prolonged blowing expiration, which was made so much of in connection with the early diagnosis of phthisis, is frequently heard in this region, and still more often in the supra-clavicular, simply owing to conduction and quite apart from any lesion. Any consolidation or thickening will of course bring it about or add to its loudness; I do not wish you therefore to discard this as a sign of apex disease. But in connection with it you must keep an open mind, since in the majority of instances it has no significance but that of an anatomical peculiarity. Its production is mainly owing to conduction, and due to the relative size and superficial position of the bronchioles.

Bronchiolar murmur is the name you have often heard me apply to this blowing breathing. I conceive its mechanism to consist in a relative deficiency or weakness of the vesicular murmur. Vesicular murmur, as you are aware, acts as a screen interposed between the ear and the sounds derived from the deeper parts of the lung. If the vesicular murmur should be inaudible or feeble, the other sounds, such as that of the air moving within the bronchioles, will be perceived, and even the relatively feeble sound of expiration may be conducted along them from the larger tubes.

The manubrial and upper parasternal regions are auscultatory areas highly specialised as regards the conduction of tracheal and of bronchial breathing and voice. I need not detain you with further reference to this very obvious peculiarity. Phthisis should never be diagnosed from auscultation of this surface alone.

At the same time vesicular breathing may, particularly in the female and in active respiration, be

very fully heard in the parasternal region, and the tubal sounds will then be correspondingly veiled.

The supra-spinous fossa gives us a repetition of that which we have observed at the "upper front" of the chest. Its inner extremity, nay, almost its inner third, which includes the entire diameter of the pulmonary apex, is highly pleximetric for the tracheal and upper bronchial sounds, and the loudness of these sounds may extend for a distance of two or three inches from the vertebra prominens, veiling almost entirely the vesicular breathing. Here again you must carefully guard against a fallacy fraught with most serious consequences. A person with the thoracic build and with some of the outward aspects of phthisis may present this peculiarity, and yet may be entirely free from the disease.

The outer portion of the supra-spinous area is pleximetric for the vesicular murmur mainly. I shall presently refer again to this point.

Of the *vertebral areas* we may first take the *cervical*. Again you have to deal here with the tracheal sound conducted to the base of the neck and for some distance upwards. The loudness of this coarse blowing sound rather increases than lessens at the level of the larynx, and it is kept up, at the occipital region, by the pharyngeal breathing. If your stethoscope be applied yet higher, to the cranium itself, you may still hear the blowing sounds tempered by distance.

The lower dorsal spine does not present any loud blowing sound, unless there be much consolidation at that level or a pleximetric conduction of the blowing breathing from a higher level.

The interscapular spine is a region of great importance and interest over which you will be prepared to find a prevalence of the bronchial breath-sound from the neighbouring main bronchi. This is heard not only over the spinous processes but on either side of the fourth, fifth, and sixth spines over a surface the extent of which varies in each individual.

Active breathers will yield comparatively little bronchial respiration, but in others, and in nearly all bedridden subjects whose posterior pulmonary areas are not much used, the area of bronchial respiration would be large and the sound a very loud one. The voice is, of course, conducted as fully as the respiration.

On Auscultation by Conduction at a Distance from the Lung.

I must call your attention once more to the clavicle and to the spine of the scapula, the percussion of which was discussed at the end of my last lecture. When we auscultate over the outer part of the scapular spine, or of the clavicle, note that we are listening at spots far removed from the lung itself, and to merely conducted sounds. Strange to say, this is an advantage, and I constantly avail myself of it. The further away from the confusing sounds of the trachea and bronchi we auscultate, so much the better shall we judge of the auscultatory condition of the lung.

For instance, in a doubtful case where blowing breathing is heard over the inner clavicular third, by placing the stethoscope quite at the outer extremity of the supra-clavicular fossa or upon the flat end of the clavicle we may obtain evidence of good respiratory murmur. This, although it would not exclude the worse significance of any blowing breathing heard nearer the middle line, would greatly favour its interpretation as a sound of conduction. On the other hand, if vesicular murmur were inaudible, and loud blowing respiratory and voice sounds were heard at the shoulder, positive evidence would be supplied of structural disease of the apex.

This test, which I give you for what it is worth, may often be of assistance to you. Do not lose sight of it when wishing to compare the vocal resonance as well as the respiratory murmur of the two apices. I believe that its employment will enable you to turn to better diagnostic account the blowing and prolonged expiration so common at the apex, and so often perplexing to the physician.

Dr. G. V. Perez's "Mediastinal Friction Sound."

Let me bring to your notice an interesting sign, the discovery of which we owe to Dr. G. V. Perez, of Orotava, and which is intimately connected with the subject of auscultation by conduction.

The "mediastinal friction sound" is, as the name implies, audible over the upper sternal region, and its loudness leaves little doubt that part of its conduction to the ear is effected through the bony thorax. This loudness and roughness suggested to Dr. Perez that the sound was of

fibrous origin, and probably due to fibrous thickening of the mediastinal structures such as occurs in chronic mediastino-pericarditis. This is an uncommon affection, and so far as published observations go, the sound in question is also unusual. Perhaps by listening for it we may identify its presence with greater frequency, and trace its production to other fibrous deposits which are less rarely met with than the chronic thickening of the pericardium and than the fibrous adhesions of the aorta mentioned by Dr. Perez in his published descriptions,* which I would suggest your consulting.

A striking peculiarity of this sound is that it may be produced at will by the patient moving the arm in various directions, or by deep respiratory movements. It is apt to be loud, coarse, fibrous, and creaking. These characters might, of course, belong to sounds set up by any form of traction or of movement imparted to mediastinal indurations or to thickened fibrous tissues connected with the thorax; and we notice that, whether we elicit them by movements of the arms or by respiratory movements of the chest, the clavicles and the ribs in both cases participate, though in a less degree, in the movement.

I will not discuss at this place the question as to whether these peculiar sounds may not be produced in the pleura covering an emphysematous lung, or perhaps in the cartilages or joints which are the seat of movement. One point I would dwell upon, and that is their exceedingly distinct conduction along the clavicle and along the spine of the scapula, and even as far as the upper part of the humerus. In a case in which I have, through Dr. Perez's courtesy, been in a position to watch this interesting phenomenon, I have been able to hear the sound with almost unimpaired loudness at the tip of the shoulder as well as over the manubrium.

You cannot fail to come across instances of this kind if you should adopt my suggestion of examining as a matter of routine the outer clavicular and scapular regions; if then on moving the arm you should hear a creaking, you would probably

* G. V. Perez, "Mediastinal Friction," 'Brit. Med. Journ.' 1896, vol. i, p. 82; *ibid.*, vol. ii, p. 717, "On a New Auscultatory Sign in connection with Mediastinal Affections."

Cf. Thomas Harris, "Mediastinal Friction," 'Brit. Med. Journ.' 1896, vol. i, p. 245.

be also able to trace its presence in the manubrial area of the chest. Until with your help we have gathered a larger number of observations, the conclusions as to the interpretation to be put upon these sounds must remain in abeyance. Your own future investigations may largely help to elucidate their ætiology.

I will conclude these lectures with a brief reference to the methods of auscultatory percussion and of auscultatory friction. Both may be regarded as luxuries rather than as clinical requisites. Yet they are invaluable when the occasion calls for the confirmatory evidence they are capable of affording.

Auscultatory Percussion.

The history of auscultatory percussion is intimately bound up with that of the binaural stethoscope.* It was introduced by Alonzo Clark and Cammann at the same time as Cammann's stethoscope. As the use of the latter failed to become general, and was ultimately given up, the method, which is not so readily carried out with the monaural rigid stethoscope, dropped out of notice, and continued to be practised only by a few adepts.

Meanwhile various improvements were made in stethoscopy. Among the improved stethoscopes subsequently introduced may be mentioned one by Dr. D. M. Cammann, "A Modification of the Binaural Stethoscope" ('New York Medical Journal,' 1885, xli, p. 27), and a valuable instrument, "Stéthoscope Amplificateur," by the late Dr. Boudet-de Paris. These I have not at hand to show you, but you are all familiar with Bazzi-Bianchi's phonendoscope which I now pass round, and this is in its principle even more than in its construction similar to Boudet's stethoscope.

The phonendoscope contains in the shallow broad metallic chamber which serves as a chest-piece and is connected with both ears by india-rubber tubes, a thin disc, the vibrations of which intensify the vibrations transmitted from the chest. You will observe that there is an outer disc which can be applied or removed, and that to this may

be fixed a metallic stem with a small chest-knob; and in this form the instrument is used for auscultatory percussion or for auscultatory friction.

Ultimately, after a long interval, the binaural stethoscope came into fashion. Cammann and Alonzo Clark's original instrument was taken up largely, but their method remained unutilised.

Within the last few years, however, auscultatory percussion has been prominently brought forward in connection with the results of the Nauheim treatment; and this may be held to have been a misfortune both for the method and for the conclusions relating to the behaviour of the heart which have been derived from its use. As it became manifest that some of the results demonstrated by its means were almost too good for unhesitating acceptance, the method was called in question and an undeserved slur has rested upon it ever since.

In the vain hope of beating the accuracy, which is very great, of ordinary percussion thoroughly performed, the method had been put to the very severe test of determining the variations in the size of the heart. Now the unsatisfactory result which followed does not logically argue anything against the method. Above all, we should not hastily condemn it on the strength of a partial trial in a single sphere of experiment. Other regions besides the cardiac afford us an opportunity of trying what it can do.

Its mode of performance.—In one of its applications, the familiar *bell-sound test*, which I need not describe, auscultatory percussion has long been in constant use. But for ordinary purposes neither coins nor pleximeter are required; the blunt end of a pencil or of a penholder, or even the finger tip, will serve as the percussing agent.

The method is based upon the conductivity of solids and of gases for sound. Thus auscultatory percussion can determine the extent of a hollow viscus and likewise that of a solid organ.

The mechanical part of the method consists in striking the surface lightly, whilst listening for the sound transmitted from the part struck. So long as the strokes and the stethoscope are within the area of the same organ there will be identity in the character of the sounds, though their intensity may not be uniform. A complete change in the character of the sound will take place as soon as the percussion or the stethoscope is shifted beyond

* G. P. Cammann and Alonzo Clark, "A New Mode of ascertaining the Dimensions, Form, and Condition of Internal Organs by Percussion," 'New York Journal of Medicine and Surgery,' 1840, iii, 62, 96.

See also G. P. Cammann, "Self-adjusting Stethoscope," 'New York Medical Times,' 1855, iv, 140—141.

the area in question. A very light percussion blow from the tip of the finger or of the pencil will suffice in most cases.

You have obviously the choice of two methods. In the first case your chest-piece remains stationary at a given spot, and a series of strokes are applied to the parts all round. In the second it is the chest-piece which travels, whilst the percussion is steadily kept up at the centre of the organ for determination.

In both cases the object is to detect any abrupt change taking place in the sounds; and this change is generally sufficiently well marked to indicate that a boundary of the organ has been reached.

Although the second method is the more stringent one, because the strokes are all delivered at the same spot and conveyed from the surface through the same medium, the first can also be made to work well, and it has the advantage of being a quicker and simpler proceeding, the chest-piece being applied once for all at a suitable spot. To be perpetually altering the listening arrangements unsettles the ear and disturbs appreciation. I believe that the bad opinion of the method which is entertained by some may have been derived in this way. I would therefore recommend your using the much more rapid method of shifting strokes, whilst it will remain open to you to now and then shift the stethoscope also if required, or to reverse your methods.

Although auscultatory percussion is quite applicable to the study of solid organs, it is of most use in the determination of hollow viscera, as is so well shown in the case of the bell-sound. You will find it specially valuable in determining the size of the stomach, and in distinguishing the latter from an inflated colon.

For my part I have had little reason to quarrel with this method, and I constantly use it. I believe that, like most other methods, it needs to be properly applied, and that careful practice is a *sine quâ non* to efficiency in its use. Do not fail, therefore, to avail yourselves of the wards for practising it.

Those who can obtain accurate results by ordinary percussion will generally be able to manage auscultatory percussion; but in the latter we lose undoubtedly the collateral help of palpation, for auscultatory percussion is exclusively a question of hearing. The chief difficulty with it has always

been the interpretation of those partial sound effects which occur at overlapping zones, such as those of the liver and of the lung, or those of the heart and of the lung, or of the heart and of the liver.

*Auscultatory Friction.**

Some months ago, whilst working with accustomed diligence in the wards over one of my cases, Mr. A. C. Pearson found after applying auscultatory percussion, which we had practised together, that by stroking the skin with the finger he could obtain still more definite results than by tapping it. The case was one of abdominal disease with greatly distended colon, one of those cases where an almost uniform note is often yielded by ordinary percussion, whilst by means of auscultatory percussion a distinction can be drawn between the individual notes of separate portions of the gut. Thanks to this fresh device of stroking the skin whilst listening through the stethoscope, the desired result was obtained with yet greater sharpness,—no longer as a difference in tone, but as a contrast between sound and silence.

Since then Mr. Pearson has devoted some attention to the method with a view to its wider application. For my part, in addition to the interest which I felt in a discovery by one of our St. George's men, I did not fail to realise its probable clinical value as an adjunct to the methods commonly practised; and you have frequently seen me apply it in the wards.

Auscultatory friction had, however, been previously discovered and described. Professor Bianchi, whose name you probably know, was its original discoverer. As in the case of auscultatory percussion this discovery was associated with the introduction of a new stethoscope, although the method is essentially independent of any special form of instrument.

The phonendoscope was first exhibited at the International Congress in Rome in 1894, and it was there with its aid that Professor Bianchi was able to demonstrate the method of auscultatory friction.†

* Cf. 'Lancet,' Aug. 27th, 1898, p. 551. "A Note on Auscultatory Friction as an adjunct to Auscultatory Percussion in Abdominal Exploration," by William Ewart, M.D.Cantab., F.R.C.P.Lond., and A. C. Pearson, B.A. Cantab.

† The phonendoscope and Boudet's stethoscope (modified and named afresh as the "splanchnometer" by

The mode of performance.—The chest-piece of the binaural stethoscope or the chest-knob of the phonendoscope is to be placed at the centre of the presenting area of the organ for investigation—the gastric area, for instance. In connection with the latter let me remind you that in order to be quite sure that you are not mistaking for the stomach some other gaseous collection—in the colon, in the small intestine, or in the peritoneum—you should invariably make a starting-point for your determination within Traube's semicircular area, because the tympanitic note of this semicircular area, which rises above the costal arch, being in all cases derived from the stomach, identifies the organ percussed as being the stomach. The pulp of the finger, or the edge of the nail, or a pencil is now lightly drawn across the skin, *radially from the foot of the chest-piece*. Although the friction is so slight as to be quite inaudible to those standing by, it is heard very loudly through the stethoscope; and so long as the finger is moving within the area of the organ it remains practically unchanged in its tone and in its loudness. As soon as the finger passes beyond the limit of the organ the sound will completely cease, or if the friction be too forcible it may still be heard, but with much reduced intensity and altered character.

It need hardly be mentioned that the friction sound elicited over a gaseous collection, and that elicited over a fluid collection or a solid organ, are of a different quality. Thus if the size of the stomach be determined by this method whilst it contains air only, its rounded contour will be made out completely; but after the subject has taken a long draught of fluid the line of fluid will intersect at the bottom of the stomach the previous outline due to the air vibrations, and this part of the outline will now be straight instead of curved. The amount of the fluid and the space occupied by the gas can be determined in succession in two separate experiments by means of the vibrations belonging to the gas and to the fluid respectively.

The alleged capabilities of the method.—The points which have just been mentioned were

practically those which Mr. Pearson made out for himself in the Williams ward. Other observers, however, had previously made an extensive study of the capabilities of the method. The scope of the latter, it was alleged, was not restricted to the determination of the size of hollow organs, but it could give the position and the boundaries of an organ such as the lung, which is aerated without being a collection of gas in the ordinary sense, and it could even define the boundaries of its lobes. Again, we were led to expect that the heart and other solid organs might be mapped out with great precision. It was even contended that one cavity of the heart could be distinguished from another, and that it was possible to demonstrate the position of the septum between the ventricles; and that in the same way the size and position of the liver, of the spleen, and of the kidneys could be made out with great accuracy.

Its failures and limitations.—Had these great expectations been fully realised, the comparative neglect which has befallen the method at a time when every new idea so quickly spreads, could hardly have been possible. This neglect, which is largely undeserved, is really the result of an over-sanguine advocacy. Though it falls far short of these early anticipations, the method holds a useful place among our clinical resources. But it must be acknowledged that its uncertainties are such that its results cannot always be trusted independently of collateral evidence, and that it is likely to be of service to experienced observers only.

The causes of this uncertainty are obvious. So minute a sound as that of auscultatory friction must be influenced by the slightest alterations in conduction, and with these we are constantly dealing not only in separate individuals, but even in the same subjects. The varying thickness and conducting power of the skin and the great conducting power of the thoracic bones assert themselves in every kind of proportion, and a strict delimitation of organs becomes difficult.

It must be borne in mind that the mechanical vibration imparted to the parietes by this light friction is so feeble that it can hardly strike very deep. Organs separated from the surface by ever so slight a layer of intervening tissue, as, for instance, the receding parts of the face of the heart which are covered by the pulmonary fringe—will

M.M. Capitan and Verdin) are described and discussed in the 'Comptes-Rendus de la Société de Biologie' for 1896; and Dr. Oscar Jennings also refers to the same subject in a letter to the 'Lancet,' April 18th, 1896, vol. i, p. 1096, but Dr. Cammann's stethoscope is not referred to. The same volume of the 'Comptes-Rendus' contains important references to the method itself.

have little chance of being animated by the cutaneous rub.

This decided weakness of the method explains its failure to give us the full size of organs, as Professor Bianchi himself admits in connection with the liver. The fault in question does not belong in the same measure to auscultatory percussion; in its performance, as I have already stated, the stethoscope may be either moved about or kept stationary, and the successive employment of these two modes of application is sometimes a valuable help, one checking the other. In carrying out auscultatory friction, on the other hand, the stethoscope must be placed at or near the centre of the area of the organ to be determined, and there is no alternative method.

A comparison of the results of auscultatory friction with those of auscultatory percussion leaves little room for doubting that the latter is the more generally useful method. Its greater efficiency is connected with the fact that the vibrations set up by percussing a central spot of the presenting surface travel to the extremity of the organ in every direction, and can be recognised by the ear even through intervening media.

Concluding Remarks.

I have called your attention in these lectures to three important recent methods of investigation, the deep palpation of organs, auscultatory percussion, and auscultatory friction. You will probably find them all of use in given circumstances, and none of them should be lost sight of in practice.

It may be stated, without unfairness, that auscultatory friction has a much more restricted range of applicability than auscultatory percussion, and that whilst both are exceedingly useful in abdominal cases, the latter is as a rule the more serviceable of the two. Deep palpation is an art somewhat difficult to acquire; but when mastered it can hardly be surpassed for rapidity and directness. Its employment, however, is contra-indicated in all cases where pressure is apt to cause damage or pain.

Thus whilst each of these methods has its special sphere of usefulness, they all have their limitations, and there is little chance that any of them will ever take the place of the old-fashioned percussion, the accuracy of which is so greatly raised by the employment of the pleximeter that

little remains to be desired after its efficient use. My advice to you is, therefore, that you should practice accuracy in ordinary percussion above all other methods, but that you should be ready to call to aid the other devices whenever a special indication for their use shall arise.

A mention of *radiography*, the most recent of our methods of physical examinations, cannot be entirely omitted. For the detection of foreign bodies it is invaluable. We cannot expect the same sharpness of definition in visceral disease, but its employment is indicated whenever a deep-seated aneurysm or tumour is suspected. Unfortunately the results are not often as clear as we could wish, whilst the process itself is a complicated one. Until this method has been considerably improved it will probably not succeed, even in the study of cardiac disease, in supplying us with data of greater accuracy than those which a thoroughly efficient percussion can provide; and for my own part I believe that for the purpose of comparing the variations in the size of the heart at intervals of time the latter still remains the more reliable guide.

Pyramidon as an Analgesic.—Landenheimer ('*Therapeutische Monatshefte*,' April, 1898) has experimented with pyramidon, a derivative of antipyrin, upon more than 100 persons suffering from some disturbance of nervous function. The headaches, which so often occur during convalescence from various psychoses, almost all yielded to pyramidon in doses of ten or fifteen grains. From one half to two hours were required for the medicine to take full effect. In the rebellious headaches of alcoholism, pyramidon was given with equally good result, the effect of the drug lasting nearly or quite ten hours. In three cases of cerebral tumour the headache was also stopped, but the dose required at first was twenty-four grains three times a day. This amount was afterwards reduced to six grains a day. Painful cramps in the limbs of alcoholic patients were benefited, and one patient who took, without satisfaction, for an infra-orbital neuralgia, 120 grains of antifebrin daily, found that thirty to forty grains of pyramidon, taken at a single dose early in the morning, would give complete relief for twenty-four hours. In no instance was any toxic symptom observed.—*University Med. Mag.*

LECTURES ON DEFORMITIES CONSEQUENT UPON INJURIES OF THE BONES AND THEIR TREATMENT.

Delivered at the City Orthopædic Hospital,
June 14th, 1898,

By JOHN POLAND, F.R.C.S.

LECTURE III.

GENTLEMEN,—Firstly as to *deformities after separation of the lower epiphysis of the femur*. The character of the deformity of the knee varies according to the original nature of the separation, whether simple or compound, whether accompanied by a fracture of the epiphysis or diaphysis, and according to the displacement of the former, and especially as to whether the fragments have been allowed to remain unreduced in their locked position. M. Trélat describes a case of deformity of the knee arising from forward displacement of the epiphysis. As it had been seen by him four years after the accident, nothing was done to rectify the displacement. In other cases which have been treated or recognised the deformity may be more and more marked as the patient walks about, the epiphysis being gradually displaced off the diaphysis. The knee may assume the varus position from displacement inwards of the epiphysis. Dr. Winslow, of Baltimore, relates a case of diastasis of the lower epiphysis of the femur, causing genu valgum, which I need not describe now. Suppuration of the joint and popliteal space has occurred in one instance three years after the accident, when the epiphysis had united in its malposition on the front of the femur. The specimen from the museum of Robert Liston is now in the museum of the Royal College of Surgeons. The patient, a girl, was fourteen years old; her leg was caught between the spokes of a wheel and the epiphysis separated. After the accident the knee remained painful and swollen, and the child halted a little in walking, but nothing beyond that ensued for three years after her apparent recovery from the accident, when a large abscess formed in the ham and lower part of the thigh, and communicated with the knee-joint. For this the limb was amputated, and the

patient recovered. In this specimen the epiphysis had been displaced forwards and upwards about halfway on to the front of the femur, and the diaphysis forced downwards and backwards. In this position a firm and smooth union had taken place with very little shortening or distortion of the limb. The presence of the end of the diaphysis in the popliteal space may also, by pressure on the popliteal artery, cause an aneurysm to develop in after years from ulceration or rupture. In the museum of the Royal College of Surgeons, Edinburgh, there is a specimen of separation of this epiphysis united in its displaced position. A beautiful illustration is given of the same specimen by Sir Charles Bell in his 'Observations on Injuries of the Spine and Thigh-bone.'

Ankylosis of the knee-joint—Some stiffness of the joint may be expected after separation of the lower epiphysis of the femur, even though the displacement has been corrected and an otherwise satisfactory recovery takes place from the injury.

Arrest of growth of the femur after epiphysal separation.—Many instances of separation have been recorded which have not been followed by arrest of growth when examined some time after the accident; so we must only consider that this is an unusual occurrence. Although bony union may have taken place, it is always to be feared that the growth in length of the femur may be subsequently interfered with by the premature ossification or permanent interference with the function of the epiphysal junction of the cartilage consequent upon the injury. The proper proliferation of the cartilage at the lower end is mostly responsible for the growth in length of the bone. The cases alluded to by Professor Humphry, König, Paschen, and others show that very grave disturbances of the growth of the tibia follow resection of the knee-joint when the epiphysal cartilage of either bone has been removed. Uffelmann was the first to direct attention to these arrests of growth. Turgis mentions the case of a boy ten years of age who had been injured by machinery. Amongst other injuries received there was separation of the lower epiphysis of the femur. When seen after the lapse of twelve years the thigh was shortened 3 centimetres. M. Delens gives a most accurate and detailed account of a case which he had observed and taken measurements of during ten years.

It was a case of compound separation of the lower epiphysis of the femur with protrusion of the diaphysis, in which the lower end was resected on account of the impossibility of reduction. A child aged eight and a half, when climbing behind a carriage in motion, had caught his leg between the spokes of the wheel. Resection of the protruding lower end of the femur was performed, and when the patient was seen ten years after the accident he was a robust young man, and came to ask if he could enlist as a soldier. From his gait it was impossible to perceive that he had undergone such a severe operation; there was only a slight dragging of the leg. On measurement, the left side was 89.5 centimetres and the right 80 centimetres, giving 9½ centimetres shortening during ten years. The sound limb had increased 24 centimetres and the resected limb 17½ centimetres. The difference was entirely in the femora, for the tibiæ had exactly the same length, 38 centimetres. The growth of the right femur had taken place mostly through the upper epiphysial cartilage, the lesion of the lower epiphysial cartilage having only produced a decrease of 6 centimetres in the total length of the bone.

A case of arrest of growth of the femur with ankylosis of the knee following injury to the lower epiphysis is recorded by Professor Nicoladoni. Gradual obliteration of the epiphysial cartilage from behind forwards ensued as the result of the injury.

The following unique instance of arrest of growth of the femur for more than three inches and deformity of the knee, the result of injury to the lower epiphysis, is now under my care at this hospital. The knee was in the varus position. A girl aged eleven came with a somewhat indefinite history of injury, namely, that the mother had placed the child in the country under the charge of some friends, and that on its return it was noticed to limp. This lameness had gradually increased up to the present time. She had been to more than one hospital in London for treatment, and nothing certain appears to have been detected; at any rate, no advice was offered as to the remarkable condition. She had not worn any boot or other appliance to correct the deformity which now existed in the right lower extremity. The whole limb was shortened for four inches, and was much smaller than the left. When she stood

upright the right knee was thrown outwards into the varus position, the pelvis tilted downwards on this side, and the spine much curved, as you will see by these photographs. On placing the spine and pelvis in its normal position by raising the foot four inches, it was discovered that the femur was rather more than three inches and the tibia rather less than one inch shorter than the left. The knee-joint could be freely flexed and extended, and considerable movement was permitted in a lateral direction by abducting and adducting the leg. During abduction a very considerable gap could be seen and felt on the inner side of the knee, between the internal condyle of the femur and the inner tuberosity of the tibia. This was due to the slightly oblique direction of the lower epiphysis of the femur and its ill-developed condition. The external condyle was more prominent than the inner condyle, and projected downwards lower. This position of the epiphysis had probably been brought about by the upper pressure of the tibia upon the internal condyle during progression. The upward displacement of the internal condyle was a very notable feature, especially when the limb was adducted and the inner tuberosity placed in contact with the inner condyle, and the shortening of the limb corrected. The limb now could be placed across the front of the left so that the right knee was just above the left. The wasting and smallness of the tibia on the affected side was probably the result of the small amount of use which had been made of this limb. The hip-joints were normal, and the shaft of the femur, especially its lower end, was felt to be small and atrophied as compared with the opposite side; and between the internal condyle and the diaphysis a concavity could be felt. The skiagrams which I show you reveal the fact that the lower end of the diaphysis of the femur was almost one inch less than the left in width just above the epiphysial line, and that the chief atrophy of the diaphysial end as seen in lateral view was in the antero-posterior direction. The skiagraphic view of the leg placed almost in a line with the thigh, displays the moderate gap in this portion between the internal condyle and the inner tuberosity of the tibia. The femoral diaphysis is also seen to be curved forwards and outwards at its lower third. Supra-condyloid osteotomy was performed with the result I now show you. [The patient was shown.]

The gap left on the inner side of the divided femur is filled up with new bone, and the patient now wears a mechanical support for her shortened though straight limb.

Deformity of the leg after separation of the upper epiphysis of the tibia.—Ankylosis of the knee and fixation of the tibial epiphysis to the femur have been observed, but are not of common occurrence. As to deformity, it has been stated that if the separation has been undetected, the epiphysis may unite with displacement inwards and inversion of the knee-joint, but that this valgus condition may result from rotation of the leg and foot without any lateral displacement. M. Rognetta alludes to a case observed in 1625 by the celebrated Séverinus. In this case twisting inwards of the knee took place in consequence of the displacement of the upper epiphysis of the tibia. Drs. Fisher and Hirschfeld have recorded a case in which there occurred separation of the lower epiphysis of the right femur, the upper epiphysis of the left tibia, and both epiphyses of the left fibula, exactly at the epiphysial line. The epiphysis of the tibia was displaced forwards and firmly united again, covering over the upper part of the tibia like a cap. Amputation had been performed at the eleventh week through the upper part of the thigh, for supuration of the knee and ankle-joints and necrosis of the fibula. The patient was a lad aged seventeen, who was injured by being caught in the driving shaft of a threshing machine and violently twisted round by it. Recovery took place. The same deformity of the upper end of the tibia after epiphysial separation exists in a specimen recently added to the Pathological Museum of St. Bartholomew's Hospital.

Arrest of growth of the tibia and deformity of the leg after separation of the upper epiphysis of the tibia may take place, but the lower epiphysis has also a share in the growth of the tibia, the amount contributed by each not yet being determined. It appears from experiments of Vogt that increase in the length of the tibia takes place mostly through the upper epiphysial cartilage, and this is borne out by the frequency with which arrest of growth of the limb is met with in disease about this epiphysis. The comparatively rapid growth of the upper end of the tibia in amputation of the leg in childhood often produces a conical stump, and necessitates the removal of the end of

the rapidly increasing shaft. Bryant figures a well-marked case of arrest of growth of the bone for one inch, with bowing of the fibula following injury to the upper epiphysis two years before. At the time of observation the child was eight years old, and the growth of the fibula continued unchecked, and it became bowed outwards and hypertrophied.

Deformity after separation of the lower epiphysis of the tibia.—Séverinus (the Neapolitan surgeon) was the first in 1632 to allude to the deformities of the knee and foot which result from separation of the upper and lower epiphysis of the tibia. As to deformity, even after compound separation, with care there should be found little of it. For example, in a case related by H. E. Clarke, of a compound fracture of the lower tibial epiphysis, the patient left the hospital with an excellent limb, the movements of the ankle-joint being perfect, with no measurable shortening. And in a case described by R. W. Smith, which was not seen until six weeks after the accident, the displacement forwards of the diaphysis had not been reduced, and gave rise to marked deformity. The internal malleolus preserved its normal relation to the foot, but not to the leg or outer ankle, whereas in dislocation of the lower end of the tibia the normal bearings of the inner ankle to the foot would be found to be lost, while those to the leg would be preserved. The parts in the case just described had become so fixed in their abnormal position that no attempt was made to replace them. A cast of the injury is preserved in the museum of the Richmond Hospital.

Arrest of growth of the tibia and subsequent shortening of the limb with deformity after epiphysial separation have not been of common occurrence. Being one of the two parallel bones, an overgrowth of the fibula causes great deformity of the ankle and foot. The fibula appears to be lengthened and much increased in thickness, and if the injury has occurred in early childhood, becomes bowed outwards. The foot is also much inverted and acquires a varus position, the child walking on the outer side of the foot, and in extreme cases even on the outer side of the ankle. Mr. Wood exhibited, before the Pathological Society of London some years ago, a specimen of dwarfing of the tibia after separation of the lower epiphysis. Bouchut also observed an

arrest of growth in the bone after fracture of the lower epiphysis of the tibia whilst the uninjured fibula continued to grow, and produced a varus position of the foot. Mr. Davies-Colley likewise showed before a meeting of the Medical Society in 1888 the case of a girl who had come under his care in 1881. At ten years of age she was said to have fractured her left tibia and ruptured the internal lateral ligament, being treated at St. Thomas's Hospital and discharged as cured after forty-four days. When she first came under Mr. Davies-Colley's notice there was great prominence of the external malleolus, and the internal malleolus was small, flat, and high with respect to the external. There was one inch shortening, and the child suffered pain below the internal malleolus, and walked on the outer border of the foot. As the deformity was increasing Mr. Davies-Colley removed one inch of the fibula and divided the tibia subcutaneously. Five months later he operated a second time, removing a wedge from the tibia. When seen nearly three years afterwards she could walk well, but still on the outer side of her boot. Mr. William Rose alluded to another case at the same meeting, of a boy with fracture of the limb above the internal malleolus. The boy subsequently returned to the hospital with marked deformity and inversion of the foot in consequence of the growth of the fibula and arrest of growth of the tibia. Mr. Rose removed $1\frac{1}{4}$ inches of the fibula, and thus restored the symmetry of the limb. Mr. Edmund Owen has also described a case of arrest of growth of the tibia in a girl aged fourteen.

The following most interesting case of deformity and arrest of growth following separation of the lower epiphysis of the tibia came under my care in March, 1891. John M—, aged 13 years, had met with an injury to his leg five years previously, namely in 1886. He was climbing over some iron railings when he caught his foot in the top of them and fell over the opposite side, hanging by his foot. A lady passing released him and took him down. He was treated by wooden splints for some weeks, but did not bear any weight on that leg for four or five weeks. After he had been walking about he felt his ankle weak, but it was not until some time later that the ankle began to "grow out." This steadily increased, getting much worse the last twelve months. The boy

now walked very lame, setting the outer margin of the foot on the ground, the foot being in a varus position; the whole leg was much wasted, and the left calf was $2\frac{1}{4}$ inches smaller in circumference than the right. The left tibia, as you will see by these photographs, was much smaller than the right, and measured exactly one inch less. The fibula was much hypertrophied and uniformly enlarged, especially at the lower part, and was very prominent, but with no sign of fracture. There was a very slight ridge and a cavity above the epiphysal line of the tibia, but no displacement. The patient complained of great weakness of the ankle, without any particular pain; the movements of the joint were good. In April, under chloroform, I removed a wedge of bone from the fibula half an inch above the epiphysal line, and attempted to replace the foot in position. The corrected position not being maintained, about a fortnight later I divided the tibia with a saw just above the epiphysal junction, and corrected the deformity of the foot, placing it in a valgus position, and producing a depression at the seat of operation on the fibula. The limb was placed in a Croft's splint. When seen some months later the position of the foot was much improved, and the sole was flat on the ground. He was ordered to wear a thick sole to his boot, and a steel support to the inner side of his leg and ankle. When seen four years later the varus position of the foot was almost corrected, but this was said to have increased again slowly since the operation. This was borne out by careful measurement, showing that the fibula increased at a greater rate than the tibia. Notwithstanding the age of the patient, which was now nineteen, it was proposed to remove the conjugal cartilage, and therefore on August 1st, 1895, I operated upon the lower end of the fibula, and found the only remains of the conjugal cartilage 25 millimetres above the external malleolus, represented by a small nodule of cartilage at the anterior extremity of the line. The rest of the line could be traced as a thickened track through the cancellous tissue. The bone was very hard and sclerosed, as at the previous operation. The whole bone was then divided by a saw and Macewen's osteotome, and an attempt made to rupture the internal lateral ligament. The gap left in the lower end of the bone now permitted the malleolus to be gradually everted.

Mr. Mansell Moullin has recently shown at the Clinical Society a case in which impaired growth of the lower epiphysis and shaft of the tibia was consequent upon a severe strain, probably with the juxta-epiphysal lesions so well described by Ollier.

Arrest of growth of the fibula after separation of its lower epiphysis.—Should there be arrest of growth following injury to the lower epiphysis, which is the most important one of the fibula in this respect, there is little probability of its giving rise to any marked curving of the tibia, the main support of the lower extremity, unless it occurs in early childhood. If the valgus position be extreme, from the continued growth of the tibia with arrest of growth of the fibula, it may be considered necessary to perform Ollier's simple operation of removal of its lower cartilage—that is, conjugal chondrectomy. Deformity may also follow separation of the epiphysis of the second metatarsal bone, as in a case recorded by Mr. J. Mason. One case of deformity following separation of the epiphysis of the first phalanx of the great toe has also been published; but these I need not describe.

Deformities of the skull, spine, and pelvis may be due to partial arrest of growth following partial separation at an epiphysal junction. In some cases only part of the epiphysis may be injured and impaired, the rest of the cartilage escaping the inflammatory change. The hindrances to which these deformities give rise may at any time become very considerable and serious. I have elsewhere shown that not only may obliteration or premature ossification of the epiphysal cartilage after injury take place gradually from the inner to the outer side, or *vice versa*, with their attendant deformities, as in cubitus varus and valgus, but that this obliteration after injury may also gradually occur from behind forwards, as in the case of the lower end of the femur already alluded to, and give rise to remarkable deformity of the knee-joint. I think it is highly probable that many asymmetrical deformities of the trunk, such as those in the skull, pelvis, and vertebral column, are brought about by arrest of growth on one side from premature ossification or by impaired function of the epiphysal cartilage on that side, the result of injury.

Now as to *lengthening* of the long bones after separation of an epiphysis. While any direct

injury to the conjugal cartilage or its immediate vicinity may arrest the growth at this end of the bone, it has been demonstrated, both clinically and experimentally, that indirect irritation to the bone or medullary canal or periosteum at a distance from this cartilage may, if the irritation be sufficient and long-continued, produce overgrowth and hypertrophy with lengthening of the end of the bone. In the human subject certain cases of central osteitis of the tibia with quiet necrosis have produced a lengthening of 6 or 7 centimetres. There is also sometimes, though rarely, seen a lengthening of the bone after fracture of the shaft in children, produced by the same cause. May this not explain the fact that fractures of the femur in childhood so frequently unite without any shortening, although a little overlapping of the fragments has been present? May not, in these rare instances, lengthening of the femur due to the irritation of the fracture have compensated for the slight shortening due to the overlapping? However, the lengthening must be very small, and only occur during consolidation.

In concluding this portion of my subject I would urge that the after-results of all epiphysal separations should be more carefully recorded than they have been hitherto. The separations should be carefully examined and the limbs examined from time to time. Until this is done, a proper estimate cannot be made as to the after-effects in all the varying conditions.

Finally as to the treatment of deformity resulting from epiphysal injuries in children. Re-fracture in *recent* cases for deformity in epiphysal separation of the bones of the limbs, and re-setting of the bone when union is found to have taken place with some amount of deformity, and when all swelling has subsided, are grave questions. The operation should only be undertaken where the deformity is very great. The lower end of the radius more especially may require this form of treatment. From such a proceeding, however, periostitis, suppuration, or severe inflammation may result, and at a later period arrest of growth of the bone from disturbance of the epiphysal function of the epiphysal cartilage. These badly united separations must therefore be treated in this manner only in the early condition, namely two or three weeks after the injury, an anæsthetic always being administered.

Of osteoclasia by the various instruments invented for this purpose, and the methods of applying these, I will treat in detail when describing the treatment of deformities after fracture in the adult.

Resection and replacement of the diaphysial end, and removal of any irregular osseous projections in old separations accompanied with great deformity, in which the natural function of the limb is greatly impaired, have been advocated and carried out with good results, both with regard to the removal of the deformity and the restoration of the function of the limb. This operation should be attempted in all cases where the anatomy of the part permits such interference, but the precise details must vary with each case. Sometimes the displaced diaphysis appears to be ready to penetrate the skin, as at the upper end of the humerus, yet notwithstanding this the natural moulding process is often sufficient to ultimately remove even considerable deformity. Projecting portions of the bone which are left after the moulding down of the parts, and tend to cripple the limb, should be chiselled away. I have done this on many occasions, with the result of considerable restoration of function and symmetry of the limb. Vicious union in the later stages, if very extreme, may require osteotomy, as has been done at the lower end of the humerus a few times. Neglected cases, or cases which have fallen into the hands of bone-setters, when seen after the lapse of some little time, are very difficult of treatment if the fragments are still unreduced. Excessive callus, articular ankylosis at the elbow-joint, are often advanced when seen by a surgeon, and require resection of the limb.

Treatment of deformity some time after the accident.—Lateral displacement of the lower epiphysis of the femur producing genu valgum, may be corrected by osteotomy of the femur above the condyles. In a case described by F. J. Shepherd, displacement of the diaphysis outwards caused remarkable obliquity and deformity of the lower end of the femur, which interfered with the lad's progression. The accident had occurred several years before. The limb was straightened by Macewen's operation, and the patient had good use of the limb. It required considerable force, in a case described by Winslow, to break up the adhesions between the fragments four weeks after injury. The marked varus position, the knees

being two inches apart, was corrected, and at the end of four weeks the limb was found to be slightly shorter than its fellow, but the knees could be easily placed together.

Loss of function and utility of the limb after epiphysial separations.—Loss of function of a limb from stiffness of the joint or muscular atrophy, the result of prolonged fixation, usually disappears after careful and systematic movement daily carried out. The loss of function of the limb may be the result of irregular union of the displacements which have not been properly adjusted, or of the excessive formation of callus at the epiphysial line, close to the articulation. Each and all of these may considerably hamper the movements of the joint and obstruct the muscles, even after the completion of the moulding process. The crippled condition of the limb and joint may under these circumstances demand, as I have said, resection of the joint, as at the upper end of the femur, where non-union or displacement of the diaphysis is a not uncommon occurrence. Removal of the head of the bone in this instance by operation is the only procedure likely to be attended with a successful result.

Operations for deformity due to *arrest of growth*. The deformity of the leg or forearm, arising from arrest of growth of one of the bones and progressive increase in the other from injuries and other causes, have been dealt with by Ollier. When the deformity due to a lesion of one of the parallel bones is commencing, it must be treated immediately by some form of apparatus which will correct the deviation, or by incising the conjugal cartilage of the sound bone. If the deformity is of longer standing, the growth of the bone which is relatively too long may be checked by removal of one or both of the conjugal cartilages. The other bone then continuing to grow, however slightly, will correct the deformity, although the limb may be threatened. Up to the present time, in this country, surgeons have been content to resect a portion of the bone. In the choice of either of these operations, a careful consideration of the patient's age, the amount of deformity, and the relative importance as regards growth of the particular cartilage, have to be taken into consideration. On the other hand, indirect irritation of the cartilage by the introduction of a foreign body into the medullary cavity and by lacerating the periosteum at a distance

under antiseptic precautions may increase the growth in length of a long bone during its period of growth. The irritation set up need not be intense, but it must be long-continued. In the fully-developed youth or adult, subperiosteal resection of the end of the sound bone may suffice to correct the deformity.

The lower epiphysis of the ulna is subcutaneous and easily reached. As regards the radius, Ollier says it should be reached from the dorsal aspect between the extensor tendons, and that the operation is a very simple one. A small knife is introduced parallel to the cartilage, which is incised, and a second cut made obliquely to the first and a portion of the cartilage removed. This is repeated several times, until enough, either one third or one quarter of the whole, has been removed to ensure the amount of arrest of growth required to be produced. Ollier recommended, however, several partial operations repeated at intervals of some months, since such great difficulty is experienced in ascertaining exactly the amount of cartilaginous elements left in the sound limb, and therefore the requisite amount to be removed from each. The effects of the first operation should therefore be carefully watched. Some surgeons think it more advisable to wait until the full deformity of the bones has developed before performing resection of the sound diaphysis.

As to operations for deformity of the ankle and foot due to arrest of growth of the tibia, removal of an osseous wedge from the fibula without transverse division of the tibia will meet with greater success if performed after the limit of the period of the growth in length is reached than when the fibula is in an active state of development. Or the simple operation of removal of the conjugal cartilages of the fibula may be practised under antiseptic precautions, when the deformity has not existed any length of time and the period of fusion of the epiphysis with the diaphysis is not a short time after—that is, before the eighteenth or nineteenth year. Ollier has successfully corrected a varus condition produced by suppurative osteitis of the lower end of the tibia by chondrectomy. He excised both epiphysal lines of the fibula, and succeeded in arresting its growth, so that sixteen years after operation the foot had lost its deformity and the lower ends of the tibia and fibula were in normal relation. As an alternative operation for

arrest of growth of the tibia he suggests exciting growth of the conjugal cartilage by indirectly irritating it—that is, by irritation at a distance, such as by a nail penetrating the bone into the medullary cavity, or by excessive cauterisation of the surface of the tibia to produce more or less superficial necrosis. In all these conditions the irritation must be prolonged in order to produce the necessary hyperplasia of the cartilage, and the cauterisation repeated many times. In some cases Ollier has combined the two methods by excision of the cartilage and artificial irritation.

Chronic Constipation in Children; Treatment by Massage.

—Cattaneo reminds us that the dangers of chronic constipation are twofold—mechanical and resorptive. The therapy must be based upon three fundamental principles, regulation of diet, stimulation of peristalsis, and improvement of the general health, with avoidance of those hygienic errors which favour constipation. In very young infants care of diet is sufficient. For atony of the intestinal muscle strychnine has been used, and atropine when the cause is a neurosis. Electricity should be used only in the worst cases, as it is painful and cannot be borne by most children. Abdominal massage is by far the best treatment, according to the method of Heubner. Before beginning the massage a dose of castor oil or a clyster should be given to prevent old fecal masses from injuring the intestinal walls. The first séance may last three to five minutes, the later ones being gradually increased to eight or ten. After the third or fourth treatment voluntary defæcation occurs, and in two to three weeks a permanent cure is accomplished. Tonic general treatment is not necessary, only the strictest hygiene, and, in the case of rachitic or scrofulous children, cod-liver oil may be administered. Ten cured cases are reported, the children ranging from two and a half months to four years of age.

American Journ. of Obstet., August, 1898.

Diphtheria of the Vulva.—W. P. Cones reports two cases of diphtheria of the vulva. Both cases were those of children about two years old. He believes that an examination of the vulva should be made in every case of diphtheria occurring in young children.

CHAPTERS FROM THE TEACHING OF DR. G. V. POORE.

VI.

GENTLEMEN,—With regard to the condition of the body immediately after death, I may read you "Researches made at Amiens on the Remains of a Criminal," by MM. Regnard and Loye, from 'Le Progres Medical,' July, 1887.

"Up to the moment the knife fell the face maintained its natural colour, which persisted when the head was examined two seconds afterwards. The features were immobile, and eyelids widely open, the pupils moderately dilated; the mouth was firmly closed; the head presented no trace of spontaneous movement, nor of fibrillary contraction. No result was produced by presenting the finger immediately before the eye, but on touching the eyeball, or the extremities of the eyelashes, the eyelids contracted as promptly as in the living subject during the first five seconds after death. Six seconds after death this reflex could no longer be evoked. The jaws were firmly clinched and could not be separated by the strongest efforts. In no other part of the body was there a trace of similar muscular contraction. One minute after decapitation the face commenced losing colour; the mouth remained firmly closed; light produced no movement of the iris; the trunk was flexed; the carotids still ejected blood; the knee reflex could not be produced. At the end of four minutes the face was entirely exsanguine, the upper eyelids were half dropped, the mouth was still firmly closed, but the point of the finger could now be introduced between the upper and lower teeth. Peripheral excitation, as pinching the tongue or sounding in the ears, produced no effect. Irritation of the spinal cord, either cephalic or of the trunk, met with no response. The body was closely observed for twenty minutes, after which interval the autopsy was commenced. On opening the thorax the heart was found to be still beating, and on opening the pericardium regular rhythmical contractions of auricles and ventricles were observed until the twentieth-fifth minute after decapitation. The auricles continued to contract for nearly an hour. On opening the heart, the left

ventricle was firmly contracted, the right ventricle being relaxed. The left lung was emphysematous at the margins—a common condition in death by the guillotine. The right lung was very adherent. The bladder was empty. The vessels of the pia mater were moderately filled with blood. There was a considerable quantity of air in the subarachnoid space. The vessels on the pia mater, especially on the convexity of the cerebellum, were filled with blood containing air bubbles. The blood was bright red. The head was separated from the trunk at the lower part of the fourth cervical vertebra. Post-mortem rigidity did not supervene until three hours after death; the temperature, taken in the rectum, being 90° F. The rigidity appeared first in the lower extremities. Six hours after death the upper extremities remained flaccid.

Conclusions.—No sign of conscious life can be observed two seconds after decapitation. Death thus produced is painless."

With regard to the teeth being clenched, you will have noticed that a man expecting a blow clenches his teeth. When a butcher pole-axes a bullock, the axe makes a hole in the forehead, and the animal drops apparently dead instantly. Then the butcher usually puts an ordinary bamboo cane through the hole in the skull, and stirs up the medulla. You must keep very clear of the animals legs while the stirring process is going on, because the spasm is tremendous. But in this man who had been decapitated, the irritation of the spinal cord produced no movement of the head or of the trunk. The account does not say whether the urine was voided at the time of the execution, that is to say whether it was found in the clothing. That abstract I have read is taken from the 'Medical Chronicle,' and was made by Dr. Dixon Mann. Of course you must always take into consideration that the mode of death here was very exceptional. Probably where the spinal cord and vessels are not divided you would get other conditions.

There are other signs which demand our attention. One sign of death is said to be loss of translucency of the hand. If we hold the living hand up to a bright light we get a gleam of red through it, but in the dead hand that partial transparency is lost. Then there is loss of elasticity of the skin, and it has been debated

as to how much the beard grows after death. I take it that just as the heart may beat for a few minutes after sudden death, and just as the muscular irritability persists for a few hours after sudden death, so the growth of the hair may, theoretically, proceed for a few minutes after death. But I think that is all. A man may be clean shaved at the moment of death, but the loss of elasticity of the skin and the pallor of the face combined often give the impression that perhaps he had not shaved that morning, and it looks as if the beard had grown a little. I should not mention this fact were it not that many years ago I was dining with five or six military gentlemen, all men of standing in the army, and one of them, whom I knew very well, was a colonel of Engineers. These gentlemen had all been in the Crimean war, and the question arose as to the growth of hair after death. I told them what I have told you, and what I still believe to be true, and my friend the Engineer colonel turned round on me and said, "You are perfectly wrong; there is no doubt whatever that in the Crimea the beards of the soldiers grew through the collars of their great coats." Now this man was a most estimable man; he was perfectly incapable of a joke, and would not have said such a thing in a joke, nor would he tell an untruth in joke. I said, "Of course, if you can assure me that you carefully examined these soldiers yourself, that you yourself carefully examined the beard and the coat, and found the hair was growing through, I will accept your statement; you are a trained observer, and if you have examined the man so closely you cannot be wrong." It then transpired that a lieutenant told him, and a captain told the lieutenant, and somebody told the captain, who had been told by Tommy Atkins, and that is how it came about, and shows that "hearsay" is not evidence. I mention this because I think it is important to show how widespread, even among educated people, are these absurd notions. The idea of the hair growing for a few minutes after death is rational enough; but that the hair should grow through a man's great coat is quite absurd; it is not alleged to have happened even to Rip Van Winkle.

Now to sum up these early signs of death. One may remark that the early condition of the dead body is of immense importance in a medico-legal sense. The recently-dead body is first of all warm

and pliant, and that exists for a variable period; it may last apparently from one to twenty-four hours. Secondly, the body may be found warm or cold and rigid, and that is a condition which may last from ten hours to three days. Then a body may be found cold and pliant, but not putrefied. And finally there is the access of putrefaction. When you go to a body the two things you look to are the temperature and the state of rigor mortis. Taking all the circumstances into consideration you must form the best judgment you can; and again I would say, do not attempt to be precise where precision is impossible; that is a mistake which is very often made. Averages of course are very useful things, but averages are made up of extremes, and you must always take the whole of the circumstances into consideration carefully before giving an opinion.

Now, there is a case in which the early condition of the dead body was of very great importance, and the case is reported at length in the big 'Taylor.' It is the case of Gardner the sweep, and I will recite it to you:

The prisoner Gardner lived with his wife and another woman named Humbler. The wife was found dead in her bedroom, with wounds in her throat, at eight o'clock in the morning of September 15th, 1862. The nature and direction of the wounds, the position of the body, and of the weapon, as well as other circumstances, proved conclusively that this was an act of murder; and as there were no persons in the house at the time of the occurrence excepting the woman Humbler (the servant) and the prisoner Gardner, it followed that one or both must have been concerned in the act. Gardner accused the servant Humbler of having perpetrated the murder during his absence from home; but as there was no evidence against this woman, he alone was subsequently called upon to answer the charge. The facts as they bear upon the question which we are now considering are very simple. Mr. S— saw the body of deceased, a healthy, well-developed woman, aged thirty-seven, at eight o'clock in the morning. Her body was found lying on a wooden floor, covered with a flannel petticoat and a chemise. The upper limbs were cold and rigid; the face, shoulders, and chest were cold; the neck was so rigidly fixed with the trunk that the entire body was lifted up with it when the head and neck were raised. The thighs

and legs were quite cold, but there was no rigidity in these parts. The only warmth found about the body was in the lower part of the abdomen; and this obviously arose from the contents of the uterus, the deceased being in the seventh month of pregnancy. The opinion given by Mr. S— regarding the time of death before its exact bearing on the guilt of the prisoner could have been known, was that the deceased had been dead *about four hours*, certainly more than three, and that she could not have been dead so short a time as two or three hours when he first saw the body. This opinion was corroborated at the trial by another medical witness.

Without going into all those circumstances which tended to fix this crime beyond any reasonable doubt upon the man Gardner, it may be sufficient to state that the defence turned principally upon the condition of the dead body when found. It was proved that from four to eight o'clock in the morning, that is for about four hours, the prisoner was absent from home following his usual occupation as a chimney sweep. It was contended by his counsel that within this short period the body might have become cold and rigid as it was found, and, therefore, that the murder had been perpetrated by someone during his absence. On this theory the woman Humbler alone was guilty. The facts proved at the trial were, however, considered by the jury to be quite inconsistent with the innocence of the prisoner, and he was convicted of the crime. This is an excellent case as showing the importance of these early signs in fixing the time of death. The body was cold and it was stiff, and that would go to show that at eight o'clock in the morning she had been dead very likely ten hours. At any rate everybody was justified in saying that the time was more than four hours.

On a recently dead corpse, *livid spots* appear, called hypostases. They are due to the blood flowing into the most dependent veins. At death the left ventricle may be full, but after death it contracts and enters into rigor, and the arteries contract also and empty themselves of blood. You know, of course, that the early anatomists were of opinion that the arteries conveyed air, because they were always found empty. The blood accumulates in the veins and takes the line of least resistance, that is, it goes to the dependent parts which are

not pressed upon. If you look at a recently dead body, such as we find in post-mortem work, lying on its back on a table, you will find where these hypostases are. You will find them in the flank, and if you turn the body up you will find there is a very dark colouration in the nape of the neck, in the loins, in the hams, and above the heels. Those parts which are subjected to pressure are not full of blood; and you must have observed again and again a great white patch over the shoulder blades and buttocks upon which the body rests, and others on the calves of the legs, if the calves were big enough to press on the table, and on the heels. These lividities are not due to extravasation; they are not accompanied by any swellings of the parts, and if you cut into them you get no hæmorrhage and find no extravasation in the tissues. These lividities are of very great importance because coroners' juries not infrequently take them for bruises when they "view the body," and consequently come to the conclusion that because there are blue marks on the body that the person has been maltreated ante mortem. When putrefaction sets in you may get these stainings in parts other than the most dependent parts, because the accumulation of gases in the abdomen may drive the blood in various directions, and you may find the hypostases on the face and elsewhere. I remember once giving evidence at a coroner's inquest, and I had considerable difficulty in persuading the jury that these marks were not bruises. However, the coroner, who had seen the body, quite agreed with me.

Now, as rigor mortis passes off putrefaction sets in. Putrefaction, of course, is a sure sign of death; by some it is said to be the only sign of death, but that is going too far. The earliest stages of putrefaction are often marked by a greenish-blue colouration of the abdomen. Sometimes you find these greenish lines taking the course of the great venous trunks. They are due to a change in the blood pigment. Then, soon you get the generation of gas, and sometimes gas distends not only the abdomen, but the tissues generally. Sometimes you get a body exceedingly disfigured in that way. This generation of gas in the body may drive the contents of the stomach and intestines out of the mouth and anus. The pressure of gas in the abdomen has even emptied the uterus in women. The gases so formed are very varied. You get

ammoniacal gases and sometimes carburetted hydrogen, which is inflammable. It has never fallen to my lot yet to be able to demonstrate that the gases are inflammable. It is a question that interests me, and occasionally, when I have had to make a post-mortem examination on a very putrid body, particularly in the summer, I have more than once made punctures, and applied matches to see if I could ignite the gases. Still, I do not think there is any doubt that they sometimes are inflammable. It probably depends on the kind of microbe that is at work in bringing about the putrefaction of the body.

As you know, putrefaction is due to the action of micro-organisms, and the rapidity of their action depends upon circumstances. There is no doubt whatever that the causes of death influence very much the rapidity of the onset of putrefaction. Putrefaction is apt to be exceedingly quick after some of the infective fevers, including acute pneumonia. Then the access or otherwise of putrefaction depends upon the condition of the body. We know that for putrefaction to set in a certain amount of water is necessary, *i. e.* a certain amount of fluidity and moistness of the tissues. The bloated drunkard rots quicker than the lean and slippered pantaloons; that is what you might reasonably expect. Thus, it is said that people who die suddenly with their bodies full of blood are apt to decompose rapidly; and that sometimes where death has been very slow and due to exhausting disease decomposition is deferred. Again, putrefaction is usually influenced by temperature. We know that in the frozen ground of Siberia they have dug up the mammoth with the flesh upon it which has not undergone putrefaction, and how long it has been buried no one but a geologist with an imagination would venture to say; and if a body could be kept from putrefying so long by extreme cold, then it is possible for the same result to occur in much shorter intervals. The temperature and condition of the atmosphere most favorable for putrefaction is "muggy" weather at a temperature of about 70°. That, again, is a matter of common knowledge. When a body putrefies, of course it gets distended with gases, becomes discoloured, the cuticle separates, and very soon the body becomes quite unrecognisable. On the other hand, if the death took place in a perfectly dry place, then putrefaction properly so-called does not

set in, and the body dries up and mummifies. Some of you may have been to, and others have read of, the waterless parts of the world, where you find dead animals which die on the travellers' routes; they are simply shrivelled up and mummified. Indeed, it is highly probable that in Egypt the practice of making mummies was simply going in the direction that Nature was going in; a person dying in the waterless regions of Egypt could only be mummified, and the people took up the natural process and converted it into an art. The proper disposal of the dead in rainless districts except by mummification must be a very difficult matter.

Considering the conditions surrounding the body, the disease of which the person died, the temperature, the dryness of the air, and so forth, you will readily conceive that it is difficult to lay down any rules with regard to putrefaction. Sometimes it is very rapid, sometimes it is very much delayed. Here, again, you must use your common sense, and form a judgment by reasoning *pro* and *con*.

NOTES, &c.

Practical Measures in Obstetrical Emergencies. — Marx in the 'New York Medical Journal,' says that many men speak of frequent post-partum hæmorrhages. He thinks these either denote faulty technique on their part, or else they call every bleeding a true post-partum hæmorrhage. He speaks of these hæmorrhages as those where in a few moments from perfect health, in good spirits, the woman lies cold, collapsed, gasping for breath, with sighing, yawning, and all those symptoms which we all recognise too well as soul-stirring and marrow-freezing. There may be no external hæmorrhage, but the large, relaxed, boggy uterus tells the story but too well. Quick, precise action is required. No theoretical measures are to be thought of. Means that have stood the test of time must be used, and used at once, to bring on firm and good uterine contraction. The writer has thrown aside everything but one of two measures. He countenances but one hot intra-uterine douche, and if this procedure does not

bring about the desired result, he does not use the irrelevant and dangerous measures, such as direct compression, ice, persulphate of iron, lemon, vinegar, etc., *ad infinitum*, but proceeds to pack the uterus with gauze, towelling, or anything he has on hand. He never goes to a case without five yards of gauze being on hand. This is a surgical means of controlling hæmorrhage. The technique of gauze tamponade is simple; one hand over the uterus, while with the other the gauze is shoveled in, as it were, until no more can be introduced. So long as this gauze remains, bleeding cannot occur, for it acts mechanically in controlling the bleeding and actively stimulates the uterus to contraction.

The after treatment is simple. Postural treatment and stimulation by the needle, with large doses of strychnine given at short intervals; infusion of a saline solution, for the heart needs a fluid, not necessarily blood, to act upon. Intravenous transfusion is difficult of application in those cases, for the veins are so small, so collapsed, that to find them is not only difficult, but valuable time is lost. Hypodermoclysis is all right if the needle and Davidson syringe are at hand, when a pint may be injected under each breast. But we have in the colon an avenue which greedily absorbs about all the fluid we can inject. The tube of a fountain syringe is slowly wormed two feet into the bowel and the salt water allowed to run in, at the same time elevating the buttocks to allow the force of gravity to act in getting in the fluid higher and higher. It is remarkable how much fluid a colon will absorb under these conditions, and how little is expelled. The author had one case where one pint was injected every hour for twenty-four hours with most brilliant results. The water should be hot, and it would not be amiss to add to the salt solution strong coffee, or liberal doses of cognac or whisky. Ergot is of little value in these cases when given by mouth, for Hemmeter has shown that it takes at least a quarter of an hour to act. As an adjuvant, ergot ("ergot aseptic") might be given hypodermically, but deep into the outer side of the thigh.

Hæmorrhage from the cervix, while not so fatal in its immediate action, can in a relatively short time exsanguinate a patient. Its causes and prophylaxis do not enter into discussion in this paper. The diagnosis is simple enough, if in the presence

of a well-contracted uterus hæmorrhage from the vagina and vulva can be excluded. Its treatment is self-evident, but by what means? Powerful traction from below by bullet forceps or pressure from above, both causing an artificial prolapse of the organ, has in his hands, by putting the uterine arteries on the stretch, caused a cessation of the hæmorrhage. Direct pressure for ten minutes, the thumb and index-finger of one hand directly grasping the angle of the tear, has answered in others; or, to the same end, clamps inserted well above the angle of laceration. Further surgical measures would be the firm utero-vaginal tamponade. These are the varieties of treatment when direct suture and needle are not on hand. The writer only advocates primary trachelorrhaphy in the presence of hæmorrhage, and not, as many have advocated, in all cases of laceration. The universal application of sutures is condemned for the reason that if the accoucheur has been surgically clean, deep tears will in the largest number of cases heal spontaneously. If the rent is sewed up, and strict cleanliness is not observed, sepsis will arise, and union not occur. The author speaks of a case where the physician sewed so thoroughly that the entire uterine canal was closed, and not a drop of lochia could escape. But in the presence of cervical hæmorrhage we recognise the only condition for the primary operation. In itself the operation is simple. Place the woman on the back, artificially prolapsing the uterus by direct pressure, or pulling down the cervix to and through the vulva—in other words, delivering the cervix into the world—passing as many sutures as are required, and tying them tightly; for, since we are operating upon a uterus which will rapidly involute, in which the parts are congested and swollen, bleeding might occur, or the wound gape from ligatures that in this wise become loose from not being tied tightly enough.

Following this technique, it is as easy to sew up a rent cervix as it is to operate upon the peritoneum. Hæmorrhage from the vagina is rare, and requires simple suturing. Spouting from the clitoris, while apparently a simple matter to treat, is one which has puzzled the writer in finding the source of the hæmorrhage. Direct pressure, or a suture passed beneath the bleeding vessel, will readily control the condition.

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SOME CASES OF INTEREST IN DISEASES OF THE BREAST.

BY

A. MARMADUKE SHEILD, M.B., F.R.C.S.

THE following cases have occurred in my practice during the last six months. They present many points of interest, especially illustrating difficulties in the diagnosis of mammary disease.

Compound Cystic Growths of the Mamma.

A woman æt. 63 had noticed a tumour of the right breast for about two years. She had had occasional attacks of pain described as shooting or stabbing. There had been no discharge from the nipple, and her general health had not been affected. Her mother died of cancer of the mamma. She had suckled a numerous family with the affected breast, and had suffered from abscess many years before. The tumour was about the size of an orange, and situated in the outer part of the breast. The nipple was not retracted, but there was a slight dimple in the skin. The axillary glands were not to be felt. The tumour was movable on the parts beneath, it felt hard and heavy, but on careful palpation a sense of elasticity could plainly be detected at one spot.

On January 18th an exploratory incision was made, and a jet of bloody fluid escaped. The tumour was a compound cyst; there was one large cyst and two smaller ones. A soft, cauliflower-like growth sprouted from the walls of the larger cyst, and nearly filled the smaller ones. The whole mamma and axillary glands were removed, as there were doubts whether this solid growth was entirely confined to the walls of the cyst or not. She made an uneventful recovery.

A careful after-examination of the specimen showed the cysts to be formed by very distinct walls. The lining membrane of them was somewhat fibrous and reticulated. The smaller cysts communicated with the larger one. They were so

filled with growth that they practically formed distinct solid tumours, the cystic character being only apparent on close examination. The remainder of the mamma was perfectly healthy. The growth was soft, vascular, and very friable; it mainly originated from the larger cyst wall, springing from a base the size of a florin. Under the microscope it consisted of intricate masses of dendritic growths, consisting of flimsy glandular epithelium growing upon a central axis or stalk. The attachment to the wall of the cyst was deep, and the papillary processes unusually long and soft, so that they became detached on washing the specimen in water.

The mammary tissue outside the wall of the larger cyst was not infiltrated by the growth, which

The nipple skin and glands were apparently unaffected.

On January 18th an exploratory incision was made and a large cyst was opened. A quantity of dark brownish fluid sparkling with cholesterine escaped. There was no trace of solid growth. The cyst was dissected out with some difficulty, and the deep wound well brought together by deep and superficial sutures. The sac of the cyst was lined by flattened epithelium; it was perfectly smooth, and was nowhere thickened as though by growth. The breast substance round seemed healthy. From the situation of the cyst it was highly probable that it originated in one of the larger ducts. This opinion was strengthened by the fact that the exterior of the cyst was white and

Diagram of compound cyst of the breast, containing papillomatous growth.

seemed to spring quite from the lining membrane. The tumour, therefore, consisted of a simple papilloma. The diagram shows very accurately the naked-eye appearance of this curious cystic tumour.

The next case is of an equally interesting nature, though the treatment did not present so much difficulty.

Large Single Cyst of the Mamma.

A single woman aged 40, a cook, had noticed a tumour in the left breast for three weeks. Six months before she had a blow upon the mamma. There was no discharge from the nipple, which was not retracted. The axilla was free. A swelling as large as a duck's egg existed on the inner aspect of the left mamma. It was movable and distinctly elastic, and somewhat oval in shape, commencing directly beneath the nipple and extending inwards.

glistening. Though it separated here and there easily from the surrounding mammary substance, near the nipple adhesion was so intimate that actual dissection was needful to separate the cyst from the breast tissue with which it seemed incorporated.

There are many points of interest and surgical importance regarding these two cases. The first might readily have been mistaken for cancer. The woman was sixty-two years of age; her mother had died of cancer of the mamma. The tumour itself felt heavy and hard, and above all things there was a slight dimple of the skin at one point. The feeling of elasticity on firm palpation alone made the idea of cyst feasible. On the other hand there had been no nipple discharge, which might have been expected in a cyst of this size. The tumour had increased rapidly, and was associated with

shooting and stabbing pains, which the lay mind, and many of our own profession who are not alive to the uncertainties of patients' symptoms, associate strongly with cancer. A dimple of the skin over a hard mammary tumour is one of the strongest evidences we have that the growth is cancerous. I have several times seen this symptom simulated by the puckering due to an old abscess, but this is the only occasion on which I have known it occur in connection with a cyst. It is to be explained by the collapse of a small cyst, or the shrinkage of part of the wall of a large one, whereby the adjacent tissues are depressed or otherwise altered in conformation. All such considerations show how unsafe it is to trust to any one symptom as diagnostic of cancer of the breast. The nipple was not retracted. When a hard carcinoma develops in immediate relation with the nipple, the latter is invariably puckered in, and drawn towards the growth. The absence of retraction of the nipple was therefore against cancer.

Exploratory incision at once clears up the diagnosis of cases like this. It often lands the operator in a fresh difficulty. Is he to remove the whole mamma or only the cyst? In a case like the first which we are discussing, this difficulty will be very great. Removal of the entire mamma was here carried out for the following reasons: (1) the patient was upwards of sixty years of age; (2) the amount of growth was very considerable; (3) a doubt existed in my mind as to how far the growth was confined to the interior of the cyst, and how far it had infiltrated its walls and involved the surrounding mammary tissue.

In cases where cysts and intra-cystic growth occur in the mamma the instances in which it is wise to preserve the breast are few. I have found cases in the "Notes of St. George's Hospital" where these cystic affections have returned in the scar years after the removal of the entire mamma. The return was doubtless due to a fragment of mammary tissue left behind, which again became affected with cystic degeneration. The malady, too, seems very likely to involve other parts of the mamma. Perhaps the only instance in which it would be right to preserve the mamma would be in the case of a young woman, and when a perfectly simple cyst exists, or at the most contains only a few simple papillary projections upon its walls. The correct surgery of cysts in the breast is to me full

of difficulties, and exploratory incision often presents them to the operator for the first time.

The second case is of peculiar interest, because the single isolated cyst seems clearly to have originated in a blow. If this patient had been lactating, the trouble would doubtless have originated in a galactocoele. Indeed, a cystic swelling forming in a lactating woman after an injury to the mamma will usually be a galactocoele or milk cyst. In the case we are discussing the cyst contained a large quantity of cholesterine, which indicated the presence of some fatty matter, and makes the origin in one of the larger ducts highly probable. This cyst might undoubtedly have been dealt with by simple tapping and the injection of carbolic acid. Obliteration would have occurred without a mark on the outside of the mamma. The difficulty of advising the injection treatment for these cases, is the doubt which may exist in the mind of the operator as to the exact nature of the cyst. Does carcinoma invade its walls? Is there intra-cystic growth? Is the cyst only part of a pre-existing tumour? It need hardly be said that to inject such cysts would only aggravate the malady. Something may be learned, too, from the contents of the cyst. Bloody fluid, or fluids coloured deep brown or chocolate, generally indicate intra-cystic hæmorrhage. This means the cyst is not simple, and the injection treatment is then rarely safe or applicable. If the fluid is clear or straw coloured, and the cyst entirely collapses on aspiration, injection is justifiable. The same difficulty occurs here as with hydrocele. The injection is by no means certain to cure. The cyst may again partially fill, or much induration may remain, raising alarm of tumour in the minds of the patient and surgeon. There is also a risk, unless elaborate asepsis is employed, of inducing suppuration; and this result occurs, I feel sure, through not employing a perfectly clean and new syringe, one which has never been used before for exploring abscess or other fluid swellings. For all these reasons the exploratory incision is, in cystic swellings of the mamma, generally preferable to tapping and injection.

Dermatitis Maligna of the Nipple (Paget's disease) in a very Early Stage.

In May, 1898, I saw a young married woman aged 36, the mother of four children, with a "sore

nipple" on the left side. She had not been able to suckle the last child with the left mamma. The nipple had been getting sore for about eighteen months, with sensations of burning and itching, and the exudation of a moist discharge. The nipple and areola around to a space of the size of a shilling were vivid crimson, raw looking and glazed, and covered with little white islets of epithelium. The parts were definitely thickened on being felt between the finger and thumb. The axillary glands were apparently not enlarged. The patient was shown at a meeting of the Dermatological Society on May 11th. All the members recognised the disease, and there was an unanimous opinion that the whole mamma should be removed. Partial removal by excision or caustics was not considered safe. This expression of skilled professional opinion has an important bearing on the early treatment of this important malady.

Singularly enough the patient, though a young woman, consented to do what was thought right, and the breast was therefore removed. The microscopical examination by Dr. Rolleston showed the usual signs of the disease, the epithelial proliferation being strongly marked. There was commencing proliferation of the epithelium in the larger ducts, but no carcinoma.

This is one of the earliest instances of "dermatitis maligna" I have ever seen operated upon, and it is unusual for patients to submit to the operation of removal of the entire breast for a malady which appears to them so trifling. The question must therefore naturally arise, what treatment is to be adopted when, as is common, the patient refuses complete removal? I should not hesitate under such circumstances to perform a partial operation, and destroy the area widely and deeply with the Pacquelin's cautery. Removal of the entire mamma is, however, probably safer, though cases with good results from the more limited operation are not unknown.

Œdematous Fibroma of the Mamma simulating Cyst.

In March, 1893, by the kindness of Dr. Brebner, I saw a lady, a widow aged 44, with a tumour of the left mamma. She had never had children. The history of duration of the tumour was quite dubious, and there was an obscure account of a blow as being the likely cause. The growth was

the size of an orange, and situated below the nipple, which was not retracted. The skin was quite unaffected, and the axillary glands could not be felt. The tumour gave a definite sense of elasticity, so that I made sure it was in part cystic; but in this, as will be seen, I was in error. An exploratory incision revealed a large tumour definitely encapsuled. On cutting into it it was soft and roseate in colour, and I felt quite uncertain as to its true nature and whether or not it contained any sarcomatous elements. Seeing the age of the patient I thought it wise to remove the whole mamma. The tumour was then removed from its bed. The breast was small and atrophic, the growth occupying the greater part of its centre. It was noticed that a considerable quantity of fluid drained away from the tumour into the vessel in which it lay, so that shortly it was surrounded by straw-coloured serum.

Microscopical examination revealed the fact that this tumour was an example of a very rare formation in the breast, pure fibroma. It was full of serum, soft and fluctuating, reminding one exactly of those œdematous fibromata of the uterus which so exactly simulate cystic growths, and so often deceive abdominal operators into the belief they actually contain fluid.

This short history carries its own lessons for reflection. I feel confident that any surgeon of experience who had examined this tumour before exploration would have felt sure it was cystic. The tumour fluctuated obviously, and was quite elastic. The naked-eye appearances of sarcoma were exactly simulated, and it seems impossible to avoid, acting for the safety of the patient, removing the whole mamma in such exceptional tumours.

An apparently Cystic Swelling in the Mamma proving to be of a Sarcomatous Nature.

A single lady aged 42 had noticed a swelling just above the left nipple for about four months. It was detected accidentally, and might have existed longer. Occasionally it was the site of slight throbbing pain. The nipple was not retracted, and there was no history of nipple discharge. The skin was not implicated in the least. The swelling was about the size of a pigeon's egg. It was elastic and apparently fluctuated, so that I felt very clear that it was a cyst. The same opinion had been expressed by surgeons both in Florence and at

Rome, and at the former place the injection treatment had been proposed by an eminent authority. Fearing from the age of the patient that the case might not prove quite simple, I urged the propriety of exploratory incision, the treatment as to extent of removal being determined by the appearances thus disclosed. Opposition was offered to complete removal of the mamma under any circumstances, but on my refusing to operate with "tied hands" this was at length overcome. The sequel shows the wisdom of this course. An exploratory incision revealed that the tumour was gelatinous in consistence, but quite solid. It was encapsuled, but the capsule itself was thickened and infiltrated, so I removed the whole breast very widely and freely. The tumour proved to be a myxosarcoma. The opinion was also expressed that it was a myxo-adenoma, but to my mind the appearances of sarcoma were undoubted.

This case affords another example of the great difficulty of the diagnosis of tumours without exploratory puncture or incision. The apparently fluctuating nature of this swelling might have deceived any one, and have led a surgeon to inject iodine and other irritants, the results of which would have been very detrimental to the patient. Severe inflammation would have been caused and rapid increase of the tumour produced.

Carcinoma containing a Blood Cyst.

On July 27th, 1898, I operated upon a patient aged 45, who stated she had borne a tumour in the left mamma for upwards of ten years. Whether this history was quite correct or not was dubious, but her medical man stated that she had consulted him for what he considered to be an induration deep in the mamma quite six years ago. The lady was married but childless, and there was no history of "blow" or other irritative causes about the breast. She had little or no pain. On examination the mamma was of very large size, the patient being exceedingly stout. The nipple was retracted, and above it was an ill-defined hard mass the size of a hen's egg. The skin was a little dimpled over it. These ominous signs made us certain of carcinoma. At one spot, however, towards the centre, was a distinct feeling of elasticity, which made one suspicious that the affection might be a chronic abscess with much surrounding infiltration. Exploratory incision was

decided upon, and the cancerous nature of the tumour was then clear. There was a blood cyst the size of a large nut in the centre of the tumour, and it was this which had led to the diagnosis of fluid. The breast, fasciæ, and surrounding fat were freely removed. The axilla was opened and the cellular tissue removed, but no glands could be detected. The wound left was of very large size, yet it healed with little pain or constitutional disturbance; very free drainage was employed, as is my custom in cases of large breast wounds, and elaborate care was taken to previously disinfect the skin.

The diagnosis of carcinoma of the breast in very obese subjects is surrounded by many difficulties. In the first place, it is often very difficult, if the tumour be small, to feel it at all, and the diagnosis of an inflammatory "induration" is only too readily made. It is worthy of note that the dimple or pucker of the skin is generally found over cancerous growths even in very fat subjects, yet if the tumour be deeply situated this sign may be absent. Again, a cancer in the breast of a very fat woman may be deceptively mobile, since the growth is really surrounded by a semi-fluid medium. The risk of the operation is perhaps slightly increased, and the surgeon cannot be too careful to exercise full aseptic precautions. On dissecting up the flaps the fat ought freely to be removed, yet not sufficiently to render the under surface of the skin white and bare. If large masses of fat be left, in addition to the risk of not removing infected tissue the union of the wound is seldom so satisfactory as it might otherwise have been. These operations in the very obese are always anxious, and the presence of sugar in the urine or albuminuria, with feeble action of the heart and bronchitis, increase the operative risk and responsibility of the surgeon. Contrary to the practice of many, I invariably employ drainage in mammary operations, and if the wound be large and deep, drainage is effected both in the axillary region and opposite the centre of the incision, and always through the lower flap.

Operations on Ulcerating Carcinoma.

There can be no doubt that the risk of mammary operations is notably increased when ulceration has taken place. The discharge is exceedingly septic, and contains staphylococci in abundance, some streptococci, and abundant bacillary organisms of

an indefinite nature. The surrounding skin is sodden with the worst products of sepsis. The mammary tissue in the neighbourhood of the cancer is generally infected, and abscess of the breast may co-exist with ulcerating carcinoma. These conditions have been emphasised by Mr. Lockwood, who points out the dangers from sepsis in operating upon these cases, and describes the various forms of bacilli found in fatal instances.* In ulcerating sarcomata the risk is even greater, as many of the cystic cavities are full of virulent pus. If a freshly cut surface, as made in breast operations, is contaminated by such septic fluids as described above, erysipelas is invited, and fatal sepsis may readily occur.

I will now mention two instances of operation upon cases of this nature, when elaborate care in the previous treatment led to sound and speedy healing.

The first case was a feeble lady of over 60. For two years she had borne a cancer in the right breast. It had ulcerated for two months. A foul crater the size of a five-shilling piece occupied the position of the nipple, and the surrounding skin was sodden and œdematous. The glands were apparently not infected. In the preparation of this case for operation, the surface of the ulcer was mopped three days in succession with pure carbolic acid, the surrounding skin was cleansed with soap and water, ether, and a spirituous solution of 1 in 500 biniodide of mercury.

This solution was used to sponge the operation area night and morning. At the time of operation the ulcer and parts adjacent were firmly squeezed, whereby a quantity of septic discharge came from the ducts, and was at once mopped up by pledgets of iodoform wool. I regard this as an important step. If the ducts be left full of septic fluids, the operator may readily squeeze them in removing the breast, and express some of the septic matter right on to the freshly cut surface. The Paquelin's cautery at a dull-red heat was next employed, and the whole ulcer and its edges thoroughly charred. The operation was now proceeded with, and though a very large wound was made, healing was speedy and free from fever.

The second case was also a patient of over 60,

who in addition to ulceration of the mamma bore a large abscess towards the axillary aspect of the mamma. She was ill and feverish, and obviously suffering from sepsis. I declined to operate unless this condition could be remedied or lessened. The abscess was freely opened, and washed out with a solution of 1 in 1000 biniodide of mercury. The ulcer was mopped with carbolic acid. In a week the patient was much better, the temperature was only a little above the normal at night. The operation was proceeded with, and the ulcer and the whole interior of the abscess, with the margins of the incision which had been made into it, were charred with the cautery, before any incision was made into surrounding tissues. The case did not do perfectly well. Some slight suppuration occurred, with a patch of cellulocutaneous inflammation in the exact site of the abscess. Doubtless the contamination had passed even beyond the reach of the cautery.

THE objections to the purse-string suture for fractured patella are the objections to all open operations. These are the opening of the joint and the possibility of infection. I do not wish to belittle the effects of suppuration in the knee-joint. It is most serious, resulting eventually in either ankylosis, amputation, or possibly death from septicæmia. But I think the dangers of infection are too much exaggerated. While the danger of septic infection exists, it is no more potent for cases of fracture of the patella than in any case of operation primarily aseptic. An objection which might be urged against this method itself is the dissection of the subcutaneous structures laterally to the full limit of the tears in the capsule of the knee. I do not think this objection can hold. Because if the joint is to be opened at all it is necessary to do the operation thoroughly, and a smaller opening does not admit of this. To me an important part of the operation is the removal of all the torn shreds of tissue, the damaged muscle, and the close suturing of the rents in the soft parts; the conversion, in other words, of a lacerated wound into an incised one. There is one precaution, however, to be observed—namely, to handle the tissues as little as possible, and then with instruments in preference to the fingers, and most of all to do the handling yourself.—*Haynes, New York Med. Journ.*

* 'Traumatic Infection,' p. 51.

CYSTITIS: ITS CAUSATION, DIAGNOSIS, AND TREATMENT.

BY

CAMPBELL WILLIAMS, F.R.C.S.

CYSTITIS or inflammation of the bladder is met with more frequently after the fortieth year of life, rather than before that epoch, owing to the fact that the causes which predispose to the more common or chronic variety of the condition, such as stricture, or enlarged prostate, are apt to become progressively more troublesome with advancing years. Cystitis may occur in an acute or chronic form, the former frequently subsiding into the latter. Although chronic cystitis is liable to exacerbations which may transform it into a subacute type, there is nevertheless a distinct degree in the intensity of the symptoms which serves to differentiate the two forms. The mucous membrane is the portion of the bladder primarily affected, but should the inflammation be sufficiently intense in the acute condition or frequently recurrent in the chronic, the muscular walls or even the peritoneal covering may become involved. Thus an autopsy of an acute case may show an intra-mural collection of pus, evidently due to septic absorption, with even peritonitis, more or less diffuse, or, should the abscess have burst within the cavity of the peritoneum, general septic peritonitis. Ulceration and rupture of a saccule in the chronic form, by causing extravasation of urine may lead to a similar fatal termination.

The causes which *predispose* to cystitis may exist either extra- or intra-vesically. To explain this, take the instance of a case of stricture of the bulbous portion of the urethra which is gradually contracting and causing a compensatory hypertrophy of the vesical muscles to overcome the increased resistance to the outward flow of urine. The cavity of the viscus becomes less and less capable of retaining the normal amount of fluid, and the calls to micturition become consequently more frequent. Now physiologists tell us that experiments show that when a muscle contracts its arterioles dilate, and more blood is furnished to the part. As a pathological example I will quote the hyperæmia produced in the eyelids of hyper-

metropic subjects. In them the oft recurring efforts of accommodation necessitate a contraction of the pupil. With each muscular effort the blood vessels dilate, and as the arterioles which supply the lids come from the same source as those affected by accommodation, they consequently share in the vaso-motor disturbance and produce redness of the lids. Theoretically, the same should happen to the mucous membrane of a frequently contracting bladder, which by becoming congested and irritable is thereby predisposed to cystitis.

Now, compensatory hypertrophy of the vesical muscles can only reach a certain point, and when in the struggle for power that limit is reached degenerative changes necessarily ensue—dilatation, sacculation, atrophy, atony, retention.

Sacculation, when only the mucous membrane and not the whole thickness of the bladder wall is extended, is probably a gradual process. Bit by bit the mucous covering is bulged out between the muscular fasciculi, but should distension occur either from voluntary retention or from sudden congestive closure of the stricture, then the sacculation becomes more marked and, even though the obstruction to the outflow be relieved, the contractive power of the viscus fails to empty the bladder. The retained urine sooner or later decomposes and cystitis results.

One often sees cystitis occurring in stricture cases who have apparently never had retention or been subjected to instrumentation.

The question is how does it arise? Are the organisms of putrefaction brought to the bladder by the blood-stream, or do they gain access from without? Many stricture patients are the subjects of chronic gleet, and it has been supposed that the urethral discharge becomes infected by organisms which by spreading up the muco-purulent tract eventually reach the interior of the bladder. The condition of the urethra behind a stricture, namely, congestion, owing probably to the oft recurring forcible dilatation of its walls, is favourable to the spread of inflammation backwards to the bladder. In the great bulk of cases infection takes place along the urethra. But that infection of distant parts by the blood stream is possible is shown by the following experiment. The periosteum of the legs of two animals has been injured by bruising; one is let alone whilst the other is hypodermically

injected at a site far distant from the seat of injury with fluid containing the organisms of suppuration, and develops *acute necrosis*, whilst the non-injected animal *does not*. Here the germs are carried by the circulation to a part where vitality is impaired. The presence of an urethral stricture and likewise an hypertrophying prostate may be said to predispose, from the very fact of their existence, to *retention*. By the term *retention* I mean that the power of voiding the urine is in abeyance, owing to obstruction, such as one sees in cases of congestive closure of a stricture, or in sudden swelling of an already enlarged prostate in the elderly after worshipping at the shrines of Bacchus and Venus. There is a distinct difference between this state and that of *residual urine*, when a bladder *partly* but *imperfectly* empties itself, as in the dribbling *overflow*, from want of contractile power, whether of muscular or nervous origin, or in *prostatic stasis* where the residuum corresponds in amount to the size of the pouch below the vesical outlet.

But does *retention* cause cystitis *per se*, that is if the retained urine is free from organisms capable of producing *inflammation* or putrefaction, or only set up, as it were, a mechanical *erythema*? The evidence we possess that *healthy urine*, if kept in sterilised vessels free from contact with air, will remain unchanged, would lead us to the conclusion that other agencies are requisite for its production. Moreover, a bladder may have contained *residual urine* for a considerable period without cystitis developing until *after* catheterization has been employed. Needless to say that this is due to want of surgical cleanliness in the instrument *before usage*, seldom to the fact that it becomes infected in its passage along the urethra. But that infection does occur in this manner can be exemplified in cases of retention of gonorrhœal origin, where the bladder becomes implicated directly after instrumentation, notwithstanding that disinfection of the urethra has been attempted and scrupulous care bestowed upon the cleansing of the catheter prior to its usage. Retention certainly paves the way for subsequent inflammation by the erythema it produces. The muscular efforts of the viscus to expel the gradually increasing fluid, gaining up to a certain point in frequency, strength, and duration of contraction, must lead to an affluxion of blood to the mucous membrane;

but when the accumulated urine has set up either a state of clonic contracture or atonic failure, then one would expect that the tension exerted by it upon the capillaries of the mucous membrane would tend to blanch them, and so lower the vitality of the coat. Moreover, the ill-effects of retention do not cease immediately after its relief; and although the bladder may discharge the major portion of its contents there is usually some amount of residual urine left, which remains as a possible putrefactive medium. Stricture, when viewed in the above light, seems to have a well-founded claim as an extra-urethral predisposing cause of cystitis.

When one considers the classical symptoms of cystitis, it would seem to be superfluous to speak of making a differential diagnosis. But if one restricts the use of the term cystitis to inflammation of the *actual* surface of the *cavity* of the bladder, it will be seen that many of its cardinal signs are shared in common with inflammation of the prostate, and, moreover, of that portion of the gland that is situated in the urethra *external to the cavity of the viscus*.

Take the case of a young adult suffering from prostatitis, either the result of excessive venery, gonorrhœa, potent injections, or injudicious instrumentation. He may have great frequency of micturition, more or less painful according to his nervous organisation, supra-pubic and inguinal pain or discomfort which are difficult of localisation, and *relieved*, not *increased*, by pressure. He complains of weight, fulness or pain in the perinæum, and urination may be accompanied by strangury and tenesmus; whilst at the end of the act blood may be voided, the latter being squeezed out, as it were, from the highly congested prostate by the final muscular acts of micturition. Such cases, as well as the chronic form of the disease, are often diagnosed as cystitis—*acid cystitis*—but I contend that these cases are *extra-vesical*, and should be regarded as prostatitis. Any one who is in the habit of making topical applications to the prostate for the cure of a granular condition can note these symptoms, for although they are transient in about half an hour, they are nevertheless very distinct and distressing while they last. One must bear in mind the fact that in gonorrhœa what was primarily a local prostatic inflammation may spread within the bladder and set up a true cystitis, or even

extend up the ureters to the pelves of the kidneys and lead to a fatal termination.

The following table is an effort to classify the various factors that produce cystitis. No attempt has been made at division under predisposing or exciting headings, since many of the causes may be said to claim a place in either category.

1. Foreign bodies that have gained access from without via the urethra :

Intentional	{ Hairpins Slate pencils, &c.	} Leading to phosphatic concretions.
Accidental	{ Broken tip of catheter Wool or lint forced backwards by catheter	

2. Foreign bodies that have gained access from without not via the urethra :

{ Spicules of bone (pelvic injuries). Bullets, &c.

3. Foreign bodies that have gained access from within :

Calculi	{ Uric acid.
	{ Oxalate of lime.
	{ Phosphatic.
	{ Cystine.
	{ Xanthine.

4. The presence of putrefactive material or agents within the bladder :

Fæces — Vesico-intestinal fistula	{ Dysenteric. Tuberculous. Malignant.
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Pus	{ Vesico-vaginal fistula.	{ Simple. Tuberculous. Malignant. Trophic.
	{ Pyelitis	
	{ Ulceration of bladder	
	{ Abscess of bladder	
	{ Abscess of prostate (bursting into bladder).	

Abscesses } Neighbouring, bursting
Hydatids } into bladder.

Blood	{ Tumours	{ Malignant. Benign.
	{ Ulceration	{ Simple. Tuberculous.
	{ Traumatism	{ Malignant. Accidental.
	{ Medicinal	{ Surgical. Cantharides.
		{ Turpentine.

Urine	{ Urethral stricture	{ Sacculaton. Dilatation. Atrophy. Atony.
	{ Prostatic enlargement	
	{ Prostatic tumours	
	{ Nervous system (participation in)	

Paralysis: injury or disease.

Saprophytic organisms	{ Gonococci. Pyogenic micrococci. Bacteria termo.
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5. From disease of the nervous system :

Spinal or cerebral paralysis	{ Disease. Injury.
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6. From constitutional causes :

Gout.
Tuberculosis.
Exposure to cold and wet.

Acute cystitis results from the introduction within the bladder of saprophytic organisms through the use of unclean instruments, such as sounds, catheters, or lithotrites ; from the presence of foreign bodies, to wit, calculi or the fragments of one that has been crushed and imperfectly "evacuated ;" from the presence inside the viscus of putrefactive material ; from the extension of gonorrhœa ; from constitutional dyscrasiæ, of which gout is an example ; from exposure to cold or wet ; and lastly from medicinal remedies, such as cantharides or turpentine. To this last cause reference will be made later on.

The onset of acute cystitis is sudden, and is accompanied by more or less constitutional disturbance, the chief factors which regulate this being the age of the patient and the resistance of his vital powers to pain and loss of rest, the state of his kidneys, and the amount of septic absorption that ensues.

The calls to micturition are incessant, for as soon as a few drops of urine have collected within the irritable bladder they are expelled with considerable suffering, for the pain of the contracting inflamed viscus is intense. The facial expression of the patient during the act of urination, the clenched hand, the contorted body, the clammy perspiration bear witness to his agony, and, though a temporary relief is gained with the ejection of the contained fluid, he has barely recovered from the exhaustion or prostration of one effort before he is compelled to make another. There is distinct tenderness or even pain above the pubes, whilst pressure made with the hand in the hypogastrium either intensifies or produces this condition instead of relieving it, as it often does when due to prostatitis. The pain from the oft-recurring spasms, the straining and tenesmus, coupled with the loss of rest, soon tell upon the patient's vital powers, and if on the top of this sepsis ensues from the absorption of the products of putrefying urine, we must be prepared for collapse. Needless to say that collapse is more apt to occur in the aged, and in those whose constitutions are broken down, rather than in younger and more robust subjects. The urine, which is at first high coloured, contains leucocytes and an excess of mucus, soon becomes bloody ; and although it may not be actually offensive to the sense of smell at the moment it is passed, nevertheless there is a detectable scent,

different from the fishy odour of pyelitic urine, that warns us that it is "on the turn." If it is allowed to stand it quickly obtains the well-known ammoniacal fœtor from the decomposition of the urea into carbonate of ammonia and the formation of sulphur compounds, this result being brought about by the fermentative action of micro-organic agents. Frequently the urine is already offensive when one first sees the patient, and evidence of septic intoxication or infection may be present, and then the sufferer is within the "arc of danger" of pyelitis, nephritis or peritonitis, should they not have already commenced. I will here remark that if one has an early case, in which the symptoms give rise to any doubt as to whether the patient has cystitis or prostatitis, that it is the urine that will settle the diagnosis. In the latter complaint, unless the patient be taking drugs such as cubebs, copaiba, sandal wood, or turpentine, the urine has the natural odour when passed, and does not show any tendency to putrefy other than is normal to ordinary urine, whereas cystitic urine is either fœtid or decomposes very rapidly after its passage, notwithstanding that precautions have been taken to prevent this. In one case you have an organism present that has either sensibly effected this change, or is in the process of effecting it, before the urine is voided, whilst in the other contamination takes place after its passage. With the onset of sepsis the temperature rises, rigors occur, the tongue becomes dry and coated with brown fur, the pulse becomes rapid and weak according to the amount of collapse that ensues. Then the stage of coma may supervene, the temperature falling to subnormal, and the signs of uræmic poisoning may usher in a fatal termination.

The picture I have drawn represents the worst phase of the disease. Happily the more common termination of an attack of acute cystitis is either recovery or subsidence into the chronic form of the complaint.

Treatment.—The treatment of acute cystitis, when due to the presence of a foreign body, should primarily be directed to the removal of the exciting cause. But every case must be judged upon its own merits, and it may so happen from the condition of the patient that it is wiser to endeavour to subdue the more acute manifestations of the complaint, and clean up the bladder before resorting to operative procedure for its removal. In a case that

does not either require such surgical interference, or in one in which it is advisable to wait before employing it, the object in view should be to prevent sepsis, to relieve pain, to give rest, and thus sustain the patient's strength. It is evident that to prevent the occurrence of sepsis the urine must either be kept from putrefying, or should that have already taken place, it must be rendered innocuous by disinfection of the bladder. The means at our command for effecting this are twofold. Firstly, by the *direct* method of washing out the bladder by germicidal injections or irrigations; and secondly, by an *indirect* route, by giving medicine by the mouth, or, what is most usual, a combination of the two.

As regards washing out. The secret of success rests in the thoroughness with which a bladder is cleansed. The best way of attaining this end is, if practicable, to do it yourself. For that reason it is certainly advantageous for the patient, if his pecuniary circumstances will allow it, to be near his doctor, so that he can give close personal supervision to the case several times a day, and see that his instructions as to hip-baths, fomentations, &c., are carried out to the letter.

It must be remembered that when a bladder is acutely inflamed that the introduction of a catheter and the injection of fluid is a painful procedure, and that it resents the presence of a large number of antiseptic lotions in that they irritate and produce severe pain and strangury. My experience is that solutions of boracic acid are borne best. I use a saturated solution of boracic acid and *dilute* it with an equal portion of *hot* water that has been sterilised by boiling, so as to raise the temperature of the lotion to 99° to 100° F. After the catheter—which has been thoroughly cleansed *before* introduction—has been passed, the washing out can be effected either by means of a funnel or with a bladder syringe, either brass or india-rubber. The latter is the more convenient, and the best size is one that holds four ounces of fluid. Small quantities of the lotion, say one or two ounces, should be frequently thrown in rather than a few larger quantities. It is more efficacious in cleansing the bladder, and less painful to the patient. This manœuvre should be repeated until the fluid returning from the bladder is seen in a *glass receiver* to be quite clear and free from washings. On an average it is necessary to wash out a bladder

at least three times daily. There are a few little practical hints in connection with the management of catheters, syringes, or bottles which have a bearing on celerity, comfort, and cleanliness.

The catheter.—A soft Jaques catheter with a large eye has the advantage that it produces less distress to the patient than does the ordinary instrument, both during its passage and also when its point is within the cavity of the bladder. The drawbacks are that it is difficult to pass if any obstruction be present, and that its lumen is easily affected by compression. On the whole, soft instruments are as efficacious and less liable to cause injury than metal ones. Always make a rule of antiseptically washing and syringing through the catheter *yourself*; and should the instrument not have a *solid tip*, pay scrupulous attention to cleaning and clearing that portion which is beyond the eye. If you are certain as to the sterility of your instrument, lubrication with vaseline, sweet or almond oil will suffice; they do not irritate the urethral mucous membrane, but if there is any doubt it will be safer to employ Lund's or some other antiseptic oil. Throw away the instrument when the surface becomes the least cracked. If it is allowed to stand in carbolic lotion this soon comes to pass. Do not forget that mercurial lotions spoil one's silver catheters and roughen them. The quickest way to fill an india-rubber bladder bottle is to unscrew the stop-cock and pour in the lotion from a glass; when the top is readjusted expel any air that may remain, and turn the tap. One thus avoids pumping air inside the bladder, and thereby frightening or distressing the patient. As to the choice of lotions, one has a great variety to pick from, and the aim of all is directed to one end, namely cleansing and disinfection. As I said before, my choice for an all-round lotion is the saturated solution of boracic acid. It is very soothing and useful, but its germicidal powers are not strong. Weak solutions of perchloride of mercury, even when diluted to a strength of 1 in 12,000, seem to irritate in the acute form, but they are most useful in the chronic type when the urine is foetid. Boro-glyceride and also quinine injection (made 4 grs. to 3j of water, but used with an equal part of warm water so as to reduce the strength to 2 grs. to the ounce) are often employed. The glycerine in the boro-glyceride seems to irritate the bladder. I have found that very

weak solutions of purified "Izal" are well borne. I have used it of a strength of $\frac{1}{4}$ x to a pint of sterilized water. Other agents that may be used are solution zinci permanganitis $\frac{1}{12}$ gr. to the ounce. This must not have ordinary but distilled water added to it if it is required to be used warm. An easy way to heat it is to stand the bottle containing the lotion in hot water. Should putrefaction of the urine be present it will be found most useful, *directly after the more acute stage of the inflammation has subsided*, to thoroughly wash out the bladder with boracic acid solution, and then to throw in some iodoform emulsion, which is allowed to *remain* in the bladder. A little is expelled at each micturition but sufficient remains to act as a disinfectant by its decomposition and the liberation of free iodine. It acts in the same way as washing out with weak iodine solutions, only it is more continuous. Its sweetening effects are very rapid. The best emulsion to use is

R. Iodoform	5 to 10 grs.
Mucilage	3j.

The mucilage is better tolerated than glycerine, which seems to irritate. The easiest way to fill an ordinary $\frac{1}{2}$ ounce glass syringe is to withdraw the cork and piston *en masse*, and to pour into the barrel about 2 drachms of the iodoform emulsion. This must have been thoroughly well shaken beforehand to ensure suspension of the iodoform. The syringe is ready for use after the cork has been replaced.

Never use iodoform when you are using solutions of nitrate of silver. At the normal temperature of the body these two chemical agents form an explosive compound.

Let us now consider what other means are at our command for influencing the course of acute cystitis; (I shall refer in a separate paragraph to acute cystitis of gonorrhoeal origin) such as hip-baths, stupes, fomentations, and mouth medicines. Hot hip-baths, of a temperature of 105° to 108° F., give great relief to pain and spasm. They may be given three times daily, the patient sitting in one for ten to twenty minutes, according to his strength. Some responsible person should be in the room at the time to give assistance should faintness occur. Hot stupes or fomentations should be applied to the belly every two hours if there is much pain. Since confinement to the bedroom is absolutely

necessary in these cases, one should have no difficulty in arranging these details of treatment.

Treatment by mouth has for its object the reduction of pain, the procurement of rest, and by direct action on the state of the urine, a curative effect on the mucous membrane of the bladder. The acidity of the urine should be reduced to a minimum, or even a state of neutrality established, and at the same time one endeavours to render it aseptic.

The following mixture is useful to that end :

℞ Sodii benzoas	10 grs.
Sodii bicarb	10 „
Tinct. hyoscyamus	30 min.
Vini antimonialis	15 „
Decoct. hordei	ad. 1 oz.

T.d.s.

The *rationale* of the prescription is that the sodii benzoas acts as a urinary germicide in a similar way to pil. acid benzoic, the sod. bicarb. reduces the acidity, the hyoscyamus and the decoct. hordei are sedatives, whilst the antimony controls the inflammation. Needless to say the exhibition of antimony must be regulated by the state of the patient's heart and pulse.

In addition or separately to this the patient may be advised to drink freely of buchu tea—at least one pint during the twenty-four hours. The following is the method of making it :

℞ Buchu folia	½ lb.
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Divide into eight packets. Put one packet into an *earthenware* teapot, and add one pint of boiling water. Allow to cool, then strain and add milk.

Other drugs that influence the urine are lactic acid, salol, alpol, and I suppose I should mention a new preparation named urotropin.

Lactic acid given as acidum lacticum dilutum has been long and favourably known, and has ascribed to it the power of arresting the ammoniacal decomposition of the urine by direct action on the micro-organisms.

Salol (phenyl salicylate) is germicidal in its action, and affects the colour of the urine, causing it to darken.

Alpol is salicylic ether of *α*-naphthol. The dose is ten grains, given in cachets, three times daily. A glass of water should be drunk with each dose. It is also a destroyer of micro-organisms. Urotropin, an American preparation, is a derivative of formic aldehyde, and strong germicidal action

on the urine is claimed for it. The dosage is from fifteen to thirty grains in the twenty four hours.

New drugs are continually cropping up, but the best of results, namely a cure, can be obtained with the number we already possess.

Pain and strangury are best relieved medicinally by morphia, if there be nothing in the state of the patient's kidneys to contra-indicate its use. It can be given either hypodermically or by mouth. If the latter channel be chosen, special attention must be paid to overcome constipation. Never use old or stale preparations of morphia for fear that apomorphine may be present and induce vomiting, and thereby add yet another tax upon the patient's vital powers. The injectio morphinæ et atropinæ hypodermica has the advantage that the atropine counteracts the disagreeable after-effects of the morphia.

Hot water enemata often give temporarily great local relief. The same may be claimed for the following suppository, *but due regard must be paid to the amount of morphia the patient is taking by other channels* when prescribing it :

℞ Morphinæ hydrochloridi	¼ gr.
Ext. belladonnæ	⅓ gr.
Pulv. tragacanth	q. s.
Butyris cacao	q. s.

To make a suppository. Use one night and morning.

Operative procedures.—When acute cystitis results from the presence of a foreign body within the bladder, it stands to reason the quickest way to cure the complaint is to remove the cause. If there be nothing in the patient's state of health to make it wiser to defer operation, it should be resorted to at once. But as I said before in certain cases it may be advantageous to allay the acuter manifestations of the cystitis and sweeten the urine before operating. In the female subject dilatation of the urethra and extraction with forceps is usually practicable. In the male, evacuation by means of a large lithotritty cannula and aspirator may suffice. On the other hand, crushing may be necessary, or the bladder may have to be opened either by a median or supra-pubic cystotomy. The latter operation has many advantages over the former. It is quickly, safely, and easily performed. There is less shock and hæmorrhage, and no important structure is wounded, unless one is very

careless, and one can digitally and visually examine the bladder. It has been urged that when the bladder is not sutured that it is difficult to keep the patient dry and comfortable. I have found that by using large pads of sterilised Iceland moss enveloped in carbolic gauze bags that no discomfort arises from that source.

Acute cystitis of gonorrhœal origin is often diagnosed when really *only* prostatitis exists. The condition that results from infection of the walls of the bladder with gonococci is most serious. By extension to the kidneys it may end fatally through causing suppurative nephritis or perinephritic abscess, or it may result in intractable chronic cystitis and lead to lifelong suffering. The extension may happen naturally, or the diplococcus may be carried to the bladder during catheterisation for the relief of congestive retention. The symptoms are those of an intensified acute cystitis, with high fever and a large amount of pus in the urine, in which one finds the gonococcus in addition to the usual cocci, bacilli, and bacteria termo. I have only had an experience of three cases of acute vesical gonorrhœa. One case died from suppurative nephritis, another had a peri-nephritic abscess and remained like the third—a subject of chronic cystitis. The treatment for this condition must be a combination of drugs by the mouth and local applications that have a distinct germicidal action on the gonococcus,—alkalies, hyoscyamus, and copaiba, to render the urine bland, and salol or other drugs that have urinary antiseptic properties, to combat the specific organism. In the acute stages neither sandal wood oil nor cubebs are well borne, as they seem to increase the congestion. Locally, frequent and thorough irrigation should be practised, with solutions of permanganate of zinc $\frac{1}{12}$ gr. to the ounce, or a $\frac{1}{2}$ per cent. solution of either protargol or argonin, or failing this, a very weak solution of nitrate of silver ($\frac{1}{4}$ gr. to the ounce of distilled water). At bed-time after the bladder has been washed out with one of these silver compounds it should *next be flushed with sterilised water* and some iodoform emulsion thrown in, which must be washed out next morning before employing a silver solution. It is needless for me to remark that the injection of even very weak solutions of nitrate of silver is a most painful proceeding in acute gonorrhœal cystitis, and that one could not apply solutions of the salt in any-

thing like the curative strength that one does in cases of gonorrhœal ophthalmia unless the patient were anæsthetised. Hot baths, diluent drinks, and a slop diet are requisite adjuncts.

Acute cystitis may follow the internal administration of turpentine or cantharides. This condition has also been noted after the external application of Spanish fly, from its absorption by the skin. The inflammation may be severe and painful, but it is quickly subdued by withholding the drug. The symptoms appear soon after its exhibition and absorption, and consist of supra-pubic and perineal pain, together with frequent spasmodic and distressing urination. If the mucous membrane of the bladder is sufficiently erythematous the urine may be bloody. Sometimes at the end of micturition a few drops of pure blood dribble from the urethra, being evidently extruded by muscular compression from a highly congested prostate. I have seen a similar condition arise in a patient who was taking turpentine for the cure of psoriasis. Excluding the odour of violets produced in the urine by turpentine, there is no special symptom to point to a diagnosis if one fails to discover the fundamental cause.

The treatment consists of rest, hot baths, barley water, or buchu tea, and the *withdrawal* of the *exciting cause*.

Gouty inflammation of the bladder leads to the most variable degree of cystitis, its limits extending from either a neuralgic condition or a slight vesical irritability to that of acute cystitis. The mucous membrane of the penis may be involved in conjunction with that of the bladder, and the patient may or may not be the subject of a muco-purulent discharge. But gouty urethritis extending to and embracing the prostate and *not* involving the bladder is common. The prostatic symptoms, such as frequent micturition and supra-pubic pain, are frequently ascribed to an intra-vesical origin when that organ is *not* involved. It is the penile discharge that gives the clue. If the patient is made to micturate the first portion of the bladder's contents into one glass receiver, and to finish up in another, it will be seen that the latter is clear of discharge, which would not be the case if such came from the bladder. Moreover, such symptoms as may be thought to arise from the bladder can be rendered very acute by urethral injections, of which not one drop ever gains access to the

interior of the viscus. Should the urethral discharge be diagnosed and treated as gonorrhœal, with astringent injections and sandal-wood oil internally, the patient's condition will be made worse rather than alleviated. Although the treatment for gouty cystitis and prostatitis is practically identical, yet the differential diagnosis is a matter of future import to the patient. If it is the prostate that is the seat of the mischief, and *not* the bladder, then one must warn the subject that the slightest sexual over-indulgence hereafter may induce another attack, whereas the same is not so likely to happen when the inflammation has been purely intra-vesical. It nevertheless behoves people who have suffered from gout in that region to remember that their prostates are liable to become inflamed from trivial irritation, notwithstanding that they have hitherto escaped.

In acute gouty cystitis the onset is sudden, and the maximum intensity of the complaint is soon developed. The age of the patient, together with past or present evidences of gout in other regions, or a bygone history of the passage of "gravel" will put one on the track for diagnosis. The subjects not unfrequently complain of burning sensations in the soles of the feet or the palms of the hands. I have noted that in many cases the pain is semi-turgescent, and that an angry red areola surrounds the meatus urinarius, also that the urethral mucous membrane is highly injected, even when discharge is absent. The urethra is often painful to pressure in distinct spots as if there were localised patches of extra hyperæmia with some effusion into the corpus spongiosum. The urine is clear, unless the inflammation has been sufficiently intense to produce leucocytes, highly acid with an excessive cloud of mucus. It often contains crystals of uric acid, which act as a mechanical irritant to the bladder and the whole urethral tract. There is generally some gastric disturbance, as evidenced by the white coated tongue; the temperature is slightly raised, and the condition of the patient is one of irritability both as to temper and body. The amount of suffering varies very much, and in certain cases is quite out of proportion to the objective symptoms.

Treatment.—In this class of case rest in bed, with a slop diet and diluent drinks, is most necessary for an amelioration of the condition. It is surprising how rapidly they improve under full

doses of colchicum without any local treatment. By local treatment I mean *hot baths*, for unless the urine should become foul and necessitate attention it is the worst thing for the patient to attempt to wash out his bladder—firstly because it is unnecessary, and secondly because the irritation of the catheter and lotions intensify the inflammation. Amongst other drugs to be taken internally besides hyoscyamus, colchicum, potash, uvæ ursi, and buchu, are the more modern preparations such as piperazine or urocedin. My experience of lithia is that while it undoubtedly does good in chronic cases its administration in acute cases is apt to intensify the condition.

(To be continued.)

Brain Tumour Simulating Suboccipital Pott's Disease.—Wenhardt, 'Neurologisches Centralblatt,' June 15th, 1898, reports an interesting case of brain tumour that was supposed to be tubercular osteitis of the first cervical vertebra. There was no headache, dizziness, vomiting, or choked disc; pulse and respiration were normal, and there were no fits. The cerebellar gait exhibited by the patient was also attributed to suboccipital Pott's disease, as the observer had seen the same gait caused by this affection and relieved when the head of the patient was properly supported. While distinctive symptoms of tumour were absent, most of those usually caused by high spondylitis were present, including stiff neck, pains shooting from occiput and nucha, and atrophy of one half of the tongue. The growth was a myxochondrosarcoma, and extended as low as and involved the second cervical vertebra.—*Medicine*, September, 1898.

Whooping Cough.—W. Thornton Parker indorses Dr. Dawson's use of small and repeated doses of quinia sulphate in *solution* in this disease. He gives two, four, and six grains to the ounce, a teaspoonful every two hours. The quinine is both antiseptic and tonic. In his cases the paroxysms almost entirely ceased in thirty-six or forty-eight hours, and the recoveries were much better than by the use of the old-time remedies, as belladonna, &c.—*Amer. Journ. of Obstet.*

CHAPTERS FROM THE TEACHING OF DR. G. V. POORE.

No. VII.

GENTLEMEN,—The rapidity of onset and the quickness of putrefaction is one of those things which vary immensely. I warned you at the close of yesterday's lecture that you must take all the circumstances into consideration, and you must not attempt to be precise where precision is not possible. Now, to show you how rapid decomposition may be, I will read you this case, which is taken from Taylor's book.

A man, aged thirty-nine, was admitted into Guy's Hospital in October, 1849. He was fat, of pale complexion, and of intemperate habits. The muscles were flabby. He died suddenly after a few days, without suffering from any symptoms indicative of danger. His death took place at 10.30 p.m., the body remained in the ward until 8 a.m. the following day, the air having a temperature of from 60° to 65°. The conditions as to cooling and rigidity were not observed during the night; but when removed at the hour mentioned decomposition had already commenced. Dr. Taylor saw the body seventeen hours after death. The skin of the face and neck had then a bloated and tense appearance from the collection of gas beneath. Blue, green, and livid red discolorations were seen more or less over the whole surface, with bladders or vesicles as in the advanced putrefaction of bodies after some days' exposure in hot weather. The gases which issued in jets from every part of the skin in which a puncture was made were highly offensive. When a flame was applied to the puncture, the gas burnt suddenly, with almost explosive violence. The gas did not discolour slips of paper moistened with acetate of lead or nitrate of silver; hence neither sulphuretted nor phosphuretted hydrogen was present. It burnt like the bright carburetted hydrogen, and I believe that it was this gas mixed with other gases and vapours derived from putrefaction. When the tense skin of the scrotum was punctured a jet of carburetted hydrogen escaped, which burnt steadily, with a pale yellowish flame, for above a minute. The state of the body precluded a post-

mortem examination, which was considered unnecessary.

Now, with a fact like that before you, you must be very careful how you draw conclusions from putrefaction; you have to take all the circumstances into consideration.

The state of putrefaction of the body is of medico-legal importance, as the following case, also from Taylor, will show:

R. v. Ellen Byrne (Dublin, 1842). Prisoner was accused of murdering her husband by strangulation, suffocation, or other violence. The Byrnes were a drunken couple, and on July 1st they retired to their bedroom, where much spirits was taken to them. The deceased was heard to speak by an attendant on July 3rd and July 5th, but not afterwards. On July 6th prisoner left the room for a short time and closed the door after her. On the 7th and 8th she was seen at the door of the room. On July 9th she ordered *two* cups of tea at 10 a.m., and at 7 p.m. she suddenly called one of her sons to come and turn his father on to his back. The husband was dead and in an advanced stage of decomposition. The body was swollen, discoloured, and covered in parts by maggots. The face and neck were black, the right eye protruded, and the tongue projected between the teeth.

When did Byrne die? Mrs. Byrne asserted that she slept with him on the night between 8th and 9th July, and that he died on the night of the 8th, and it is possible that a body in a close room in July might reach an advanced stage of decomposition in twenty-four hours. On the other hand, the deceased was never seen alive after July 5th, and did the state of the body represent a four days' decomposition? There were witnesses who adopted both these theories, and both were sufficiently plausible. It is certain that Mrs. Byrne must have known of her husband's death when she called for two cups of tea on the morning of the 9th. The deceased probably died from suffocation, possibly from falling over on his face among the bedclothes while drunk, and, since there were no signs of strangulation (which might, however, have been obliterated by decomposition) and no motive for murder was alleged, Mrs. Byrne was acquitted.

Undertakers by preference place a corpse in a cold room with the windows shut. There are cer-

tain poisons which tend to preserve the body, and it is important to know this fact. Arsenic is one of these, carbolic acid is another. It stands to reason that if a person has been killed by an anti-septic poison the body will keep longer than a normal one would. Many interesting facts have been acquired of late years with regard to decomposition of the body, and M. Megnin, a member of the French Academy of Medicine, has written a very interesting book called 'La Faune des Cadavres,' and has described the insects which are largely operative in making away with dead bodies. I will tell you what his conclusions are. The majority of the bodies which Megnin studied had been left exposed to the air. M. Megnin has shown that insects do not attack the carcass of an animal in any haphazard fashion, but that the kind of insect which is found upon the corpse depends entirely upon the stage of putrefaction on which it has entered. M. Megnin says that a dead body which is exposed to the air is made away with by successive squadrons of insects. The first squadron that attacks the carcass are all Diptera, and belong to the genera *Musca*, *Curtoneura*, and *Calliphora*, i.e. the common house-fly, blow-fly, and allied species. The attack by the house-fly and the blow-fly are made before putrefaction sets in. The next squadron, which is also composed of Diptera, are attracted by the smell of commencing decomposition. These include a fly of a metallic green lustre (*Lucilia Caesar*), as well as flies of the genus *Sarcophaga*. M. Megnin says that among these flies there are specialists which prefer the flesh of particular animals. When we study entomology and bacteriology in relation to dead bodies and excreta we find specialism carried to an extraordinary degree. The third squadron appears as soon as the fats of the body begin to undergo acid fermentation, and the Diptera then give place to certain Coleoptera or beetles (genus *Dermestes*), and Lepidoptera or moths (genus *Aglossa*). M. Megnin mentions in this connection the *Dermestes lardarius*, or bacon beetle. The next change in the body is a caseous one, and when this begins the Diptera reappear. M. Megnin mentions in this connection the *Pyrophila casei*, which breeds jumpers in cheese and anthomyia. These are accompanied by a beetle (genus *Corynetes*) which is fond of rancid fluids. To the caseous fermentation succeeds ammoniacal liquefaction; and then

comes the fifth squadron of Diptera, belonging to genera *Tyreophora*, *Lonchea*, *Ophyra*, and *Phora*, and Coleoptera, Silphida, and *Necrophora*, to feed upon it. The sixth squadron consists entirely of Acari or mites, whose function is to dry up the carcass and reduce it to a mummy-like condition. Megnin figures six species of mite and mentions others. Then the body becomes attractive to the seventh squadron, which consists of beetles and some forms of moth, which are the scourges of the housewife, and a vexation to the furrier and the collector of dried specimens in museums. The eighth and last squadron consists of two species of beetle, which consume and make away with the pupa-cases, dung, and débris of the seven squadrons which have preceded them.

This is a very interesting study, and to Megnin undoubtedly belongs the credit of being the first to embark upon it. It is not a very attractive branch of natural history, but still it is a most important one, because there is great need of accurate knowledge concerning the dissolution of the body. Megnin, as an entomologist, says that by the eggs or larvæ, or other evidence that he may find in the dead body of such and such insects, he is able to tell how long the body must have been dead. He assumes that these squadrons of insects come in proper order, and as an entomologist he knows how long they take to lay their eggs, to produce their young, and so forth. On the other hand, it is but right to say that other entomologists state that the remarks of this pioneer are not quite accurate. That, again, one can understand. You will notice that the insects come attracted by the condition of the body, and I take it that the caseous change, the ammoniacal change, the acid fermentation change, and so forth, are caused by vegetable microbes, and it is only reasonable, I suppose, that the vegetable microbes which grow in the fat and watery body would be different from the vegetable microbes which grow in a dry and thin body. Thus it behoves us not to run these theories too hard. Still Megnin's researches mark a distinct advance.

When bodies are buried in moist positions there is formed a material which is known as adipocere, a fat-wax, a sort of soap formed by the union of fatty acids with the ammonia in the body. It has a peculiar smell, such a smell as you sometimes get in old anatomical preparations which have

been macerating in water. It is inflammable, and if you heat it over a spirit lamp it melts and burns with a sooty flame.

I have given you some account of the state of a body that is left above ground. Sometimes bodies which are ripe for decomposition, and which have not been previously sterilised, are put in lead coffins, and sometimes untoward accidents have happened. Decomposition may go on and perhaps burst the coffin. On the other hand, if bodies are put into lead coffins early they are preserved for long periods, and the form and features may be recognisable after a considerable interval. Bodies in this country are almost always buried in coffins, and are buried at a considerable depth. You may remember that Hamlet, when he accosted the gravedigger in the 5th Act, asks him how long will the body lie in the earth ere it rot; and the gravedigger's answer was to the effect that a body should last you some eight or nine years. It is, I believe, the evidence of sextons in the present day that if a body be buried at a considerable depth it takes eight or nine years to make away with it. I believe the rate of disappearance of a body depends very largely on the depth at which it is buried. If you bury a body without a coffin in the upper layers of the earth, which is full of microbes and burrowed by insects in every possible direction, then the body is got rid of very quickly, say in twelve months or so. In this country it is illegal to bury a corpse at a less depth than four feet, but I will tell you of an experience I have had in the exhumation of animals. The first animal we dug up was a cow that had been buried eighteen months previously, and had been buried only superficially. It was buried in Hampshire, on the chalk; a hollow had been dug in the chalk big enough to take the cow, and the earth was piled on top of that. On exhuming this cow we found nothing but the bones; they were beautifully clean. Next we went for a favourite old horse, which had been buried very near the same spot, but about a year longer; that was buried eighteen inches deeper than the cow. We found that the horse's flesh was coming off the bones; there was no disagreeable smell. Then we went in search of the carcass of a pet pony. This pony had been interred very deeply indeed. It so happened that the gardener had used the spot for making a pile of dead leaves for leaf mould.

When we got to the depth where the pony lay the pickaxe came upon something with a soft thud, and the odour sufficed to cause us to abandon the work. It was clear that decomposition had not proceeded nearly so far as in the case of the other two animals which had been buried nearer the surface. I think there can be no doubt whatever that if you want a body to disappear quickly you must bury it superficially. I have heard Sir William Flower say that many years ago a gentleman who lived on the coast of Norfolk wrote to him and said a whale had been cast ashore near his place, and that if it was wanted for the Natural History Museum it was at Professor Flower's disposal. They thereupon got it on shore and buried it near the margin of a wood or copse, and in two years' time Sir William Flower exhumed it and got a most beautiful specimen of the bones quite clean. Sir Seymour Haden has exhumed calves after a year's superficial burial, and found nothing but bones. I have buried a large number of smaller animals. I found a cat at the end of sixty-nine days was not quite clean, but with a little shake and holding it under the water-tap there was a perfect preparation of the bones. Worms do not eat carcasses if they can get anything else in the shape of leaves and vegetable refuse.

Of course, there are great difficulties in the way of superficial burial without coffins. Some people die dropsical, and you have to be sure that in burying a dead person of this kind it is done decently, and that there is no leakage of fluid. Therefore there must be some impermeable vessel to take the corpse to the grave, at all events. Another very important matter is this: if you bury bodies too superficially you may get animals trying to exhume them.

I think there can be no doubt that if you want to get the greatest amount of good and the least amount of harm from a dead body it should be buried superficially. There is no doubt that bodies so buried very soon sweeten, and they are exceedingly fertilising. If you bury an animal at the root of a shrub you will find the shrub grow, and will find the roots among the dead body, the dead body being perfectly sweet. The only way to purify the soil is to grow something. In many countries cemeteries are used over and over and over again. Two or three years after the bodies are buried the bones are taken up, and in many

large cemeteries on the Continent you will find a charnel-house where the bones are collected. By growing something over these bodies you produce wood, and I think there can be no harm in using such wood as firewood, and secondary cremation of that sort is profitable all round. But if you bury bodies deeply, like the pet pony I spoke of, and especially if you bury in a brick vault, and water flows in on the top of this putrid mass, leaking under pressure to a spring, then I think you may theoretically get very considerable danger. We hear a great deal about the evils of cemeteries. All I can say is that as far as I know there is no good evidence of serious evil. The Local Government Board publications are perfectly silent on the point, and last year I was present at an inquiry about a cemetery which was very interesting, because the witnesses who came up one after another gave no definite evidence as to the evils of cemeteries. I do not mean to say that the old city churchyards, for instance, in which there were nothing but heaps of rotten corpses, have not been the cause of ill-health, but I cannot find any evidence of epidemics arising in this way.

When persons are *found dead* we have got to determine how they came by their death. The police are informed, and the police generally call the nearest doctor, who is asked to come and look at the body. It may be that the body is under a hedge, or in a field, or in a room. The body may have died from natural causes, or from an accident, or suicide, or homicide. Especially in country places you have to remember that this duty of looking at the dead body is an important one, and that you are a common witness as well as a skilled witness. You will very likely be the only person of education who sees the body. It is very important, when you are called to a dead body, that you should know whether the body has been moved or touched before you saw it. You will generally be able to get evidence on that head. Perhaps some fussy person has moved the corpse from the position in which it originally was, and that is misleading. If you are sure it has not been moved you will take careful notice of its exact position. For instance, if a dead body were found on this floor you would make a rough plan, giving of the room, and the position of the body, door, and so on. It is very important that you should listen to everything that is

said, you should utter no hasty opinion. Keep your eyes and ears open, and your mouth shut. Look at the ground and see if there are footprints or signs of a scuffle, and examine the condition of the clothes, and so forth. I will read you a case from the book of Paris and Fonblanque which is full of instruction.

"A Cornish peasant, engaged in attending upon the lighthouse on the western coast, was found dead in a field near the public road leading from Penzance to the Land's End on Sunday, December 12th, 1813. He was lying in a dry ditch, with his stick at a little distance from him; one of his shoes was down at the heel, and both were smeared with mud; his pockets were empty. The body was taken to a public-house in the village, and the coroner having received notice of the occurrence, an inquisition was taken, and the verdict of wilful murder returned against some person or persons unknown. The body was afterwards buried, but a rumour having arisen that the anatomical inspection had not been sufficiently minute and satisfactory, it was, by an order of the magistrates, disinterred, and the author was desired to assist in the further investigation of the subject. Upon examining the body, which had not yet advanced so far in putrefaction as to obliterate the traces of violence, or to confuse the appearance they presented, patches, arising from extravasated blood, were seen in different parts of the throat, and distinct abrasions corresponding with the nails were visible; the face presented the physiognomy of a strangled man. On the chest, bruises, evidently occasioned by the pressure of the assailant's knees, were also noticed. Upon dissection the brain was found excessively turgid with blood. The rest of the organs appeared in a perfectly healthy and natural condition. It is worthy of remark that the field in which the deceased was found contained several shafts of abandoned mines. Upon visiting the spot the author observed tracks on the grass, as if it had been scraped, proceeding in a direction from the hedge next the public road to that in the opposite part of the field, and under which the body was found; near the former hedge also some fragments of a glass bottle were discovered. The deceased, it appeared, had been at Penzance for some medicine, and it was proved that he had left that town, on his return to the lighthouse, with a phial in his pocket. All

these circumstances combined placed the murder beyond conjecture. He had evidently been strangled, probably at the spot where the glass fragments were found, which were undoubtedly the remains of the phial broken during the scuffle; besides, it would appear that he had been dragged along the field from this spot to the opposite hedge, for marks denoting such an act were visible on the grass, and this received further confirmation from the condition in which the shoes of the deceased were found. Who then did the murder? From the circumstances of its having been perpetrated in a field containing several old mines, without any attempt on the part of the villain to avail himself of the advantage which these caverns would have afforded for the concealment of the dead body, the author was convinced that the perpetrator of the deed would be found in some stranger to the country, for such a one alone could be unacquainted with the mines to which we allude. The suggestion of this idea very naturally gave the direction to the line of inquiry. Were any suspicious strangers in Penzance or its neighbourhood? Had the deceased been seen in the society of any person unacquainted with the country? He had been seen, it was discovered, playing at cards in a public-house with some of the privates of the artillery stationed in the Mount's Bay, amongst whom was a very powerful and athletic Irishman of the name of Burns, who had lately landed, and immediately enlisted into the corps. Burns was accordingly arrested on suspicion, when the purse of the deceased containing thirty shillings was found on his person. He was, moreover, unable to show where he was at the time the deceased left Penzance in the evening; and he was subsequently recognised by two witnesses who had seen him accompanying the deceased on the road towards the Land's End. It is only necessary to add that he was convicted and hanged; and it is not the least satisfactory part of this case to state that on the evening previous to his execution he confessed to the author that all the circumstances of the case occurred precisely as we have stated, that he strangled his victim with a pocket handkerchief, but that from the difficulty of completing the act he was compelled to place his knees upon his chest."

That shows you how very useful a clever man, such as Dr. Paris undoubtedly was, may be in an investigation of a crime.

NERVOUS AND MENTAL DISEASES FOLLOWING PELVIC OPERATIONS.

By HAROLD N. MOYER, M.D.

THE topic assigned to me is a narrow one, namely, the nervous and mental diseases or changes which follow pelvic operations in women. The purpose of the inquiry is to show if there is any difference in the nervous and mental effects which follow operations upon the pelvic organs from operations done on other parts of the body, or surgical operations in general. It would be easy to discuss this subject from one's individual experience, but it would be exceedingly narrow. The observations that come to any one man are certainly few as compared with those that come to a large number of operators, so that I shall not relate individual cases, but will draw conclusions, so far as I can, from the literature which is accessible to me and the material which I have collected. The literature is disappointingly deficient in many respects. Reported cases of the nervous and mental changes which follow operations in general are very disappointing, especially when studied from a statistical standpoint. There have been a large number of studies made and cases collected of the nervous and mental phenomena which follow operations in general, but when we come to analyse this material we find that it is so varying expressed in different groups and in different tables that an adequate comparison of a considerable number of cases is well-nigh impossible. If you think for a moment you will see that the subject is one so complicated in its nature that it scarcely lends itself to statistical treatment at all. If you remember, for instance, the widely varying opinions as to the efficacy of a particular treatment of typhoid fever, or, indeed, so recent a subject as the antitoxin treatment of diphtheria—a disease which is comparatively the same in all individuals, being of short and very nearly the same duration—it would seem as if the statistical method would throw abundant light upon the value of these treatments, and would serve at once to settle the question as to their value. But, as a matter of fact, it has not done so conclusively in all respects. When we come to consider the subject of operations and their effects, you can see that the increased factors which we have to consider constitute such a varied problem that accurate, clear, and concise conclusions can not be drawn. I can only bring to you to-day such gener

ments and such general conclusions as I myself have arrived at, without being able to give in figures the basis upon which they rest.

The first question which naturally presents itself is this: Is there any difference between operations upon the pelvic organs of women and operations in general? If you examine the statistics you will be at once struck with the number of cases which are operated upon for pelvic disease, which show insanity, neurasthenia, and other nervous troubles following operation and probably resulting from it. Some writers have come to the conclusion that these operations are peculiarly liable to be followed by such phenomena; but if you come to analyse the cases more closely, you will find that operations are performed upon persons whose general health is greatly impaired. The great majority of them are very much reduced by a long history of illness prior to the operation. The operations in themselves are many times prolonged, and a by no means infrequent condition is the one of infection, a slow poisoning of the system for which the operation was undertaken. Those necessarily are apart from the operation itself, and they furnish a fruitful soil upon which nervous troubles and insanity are built. In order to get some sort of basis for comparison, I have taken fifty cases of operations upon the prostate and bladder. I have thought there might be some comparable relation between the pelvic organs in the male and in the female. The operation was on the same part of the body, and frequently accompanied by ill-health, and often in the male by infection. I compared fifty cases, excluding all those in which there was marked disease of the kidney, and I found that the nervous and mental effects following operations which might be attributed to the operation *per se*, were much greater in the male than in the female. Taking all factors into consideration, operations upon the female pelvic organs are not attended with more nervous disturbance, such as neurasthenia or insanity, than are operations in general surgery.

The character of the mental disturbance which may follow operations is substantially the same, whether the operations are done upon the pelvic organs or upon other parts of the body. The great bulk of them fall within the class of neurasthenia, or that condition which is described by alienists as primary confusional insanity. These

two conditions seem to be the great predominating mental and nervous states which follow operations.

Again, I tried to study the question as to the particular character of the operation, and as to the part that was involved in the operation, and to find out if that had a bearing upon the question. I am unable to offer any conclusion on this point. Those operations which include the ovary or Fallopian tube, and which are followed by the artificial or early menopause, were carefully studied, and I particularly addressed myself to the question as to whether there was any difference between the nervous phenomena of the artificial or induced menopause and the nervous disturbances which accompany the menopause at the ordinary time of life. My conclusion is that there is no essential difference and that they are practical, so far as nervous phenomena are concerned.

Another question is: Whether the removal of the ovary added an additional factor, in the sense that there was something taken out of the economy, if you please, by which there was created an unstable or peculiar nervous state; something that was comparable to the removal of the thyroid gland or an internal secretion cut off, and whether there might be mental phenomena growing out of the removal of the ovary distinct in their characteristics from the artificial menopause? I have no conclusions to offer upon that phase of the subject. I have not formed an opinion as to whether that is so or not, as the evidence is very conflicting. It may or may not be. Certainly, so far as the effects of operations are concerned and so far as my studies go, there seems to be a practical point in this, that the ovary had better be left in, or a part of it, if possible, and there is also sufficient to warrant the deduction that resection of the ovary is far preferable to removing it, if it is possible to do so.

In conclusion, therefore, I would simply say that I have arrived at the opinion that there are no radical differences in the primary effects of operations upon the pelvic organs in men differing from the effects of similar operations in women, or indeed of operations in general surgery, and that there is a distinct and peculiar effect from the removal of the ovaries or tubes, or both, by which the menopause is established, but that that does not differ in its nervous phenomena from the menopause occurring normally at the ordinary time of life. As to the possibility of a peculiar physiologic effect following extirpation of the ovary, not attributable to the menopause, I have absolutely no conclusion to offer.

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ACUTE AND CHRONIC MIDDLE EAR SUPPURATIONS: THEIR TREATMENT.

BY

PHILIP R. W. DE SANTI, F.R.C.S.,

Assistant Surgeon, Aural Surgeon and Lecturer on Aural and Minor Surgery, Westminster Hospital.

OF all the diseases of the ear that come under the notice of the aural surgeon, by far the most frequent is purulent inflammation of the middle ear.

The presence of a discharge of the ear or ears is often looked upon with complete indifference by patients or their parents, and even not infrequently by their medical advisers, and the consequences and complications are often so grave that no excuse is needed for the present article.

Not a week passes by without my seeing at the aural department at the Westminster Hospital several patients suffering from middle ear suppuration of from six months' to as many or more years' duration, and who, on inquiry, state they have adopted no treatment at all, and who have finally come, not on account of the discharge, but for increasing deafness, an attack of pain, or some possibly dangerous cerebral or bone complication.

On asking the parents of such patients, or the patients themselves, their reasons for this neglect, it is no uncommon thing to be told that they have been assured they will grow out of it, or that as long as the discharge is free no danger need be feared.

Medical men, indeed, have been known to actually recommend a course of sea-bathing for such a condition.

Purulent discharges from the middle ear are seen principally in children, and occur as an exacerbation of an existing catarrhal inflammation or in an ear previously normal.

The causes therefore of catarrhal middle ear inflammation are also those of purulent median otitis. Of such cases, by far the most frequent are

those originating in extension from a similar affection of the naso-pharynx, as from the acute exanthemata, especially scarlet fever, measles and diphtheria, adenoids, enlarged tonsils, nasal and pharyngeal catarrhs, repeated colds, influenza, &c. This ætiological factor is a matter of great importance in relation to the prevention of tympanic inflammation and in the treatment thereof when present.

Micro-organisms also play an important part in the ætiology of purulent median otitis; they find ready entrance to the middle ear by way of the Eustachian tube and external auditory meatus. In some diatheses, especially the tubercular, purulent middle ear inflammation is common, and, as a rule, obstinate to treatment and often grave in its complications.

Trauma, as from fracture of the middle fossa of the skull, the entrance of some foreign body, or the entrance of fluid through the Eustachian tube, or damage to the latter from naso-pharyngeal operations, may also give rise to purulent inflammation.

The object of this article being to deal with purulent discharges from the middle ear itself, I exclude cases of pus flowing from the external auditory meatus, due to such conditions as eczema of the canal, ulceration from foreign bodies, abscesses bursting into the canal from a diseased temporo-maxillary joint, suppurating dermoids, or sebaceous cysts. I only refer to these conditions in order to emphasise the fact that so-called otorrhœa, by which is meant purulent discharge from the middle ear, does not necessarily imply a perforation of the tympanic membrane.

I will deal with the subject under two headings—

1. The stage before perforation.
2. The stage after perforation.
 - (a) Stage of purulent discharge.
 - (b) Non-secreting stage.

1. *The Stage before Perforation.*

The symptoms present in this stage may be very severe—acute otitis media purulenta,—or on the other hand, slight and ill marked,—subacute and leading to painless perforation.

When severe they are as follows: Excruciating pain in and around the ear and head, causing the patient to scream out, and sometimes, especially in children, ending in delirium, vertigo, raised

temperature (101 to 103° F.), quick pulse, and furred tongue. Convulsions and coma occasionally occur. Deafness is more or less marked, but in the presence of the severe pain is probably not noticed. The external meatus may be swollen, red, and very tender.

In young children the intense pain in the head, the screaming, delirium, and fever may lead to a diagnosis of meningitis; the onset of the latter is, however, usually not so sudden, the pulse and respirations are different, and cerebral vomiting is absent; there are also usually no eye symptoms, such as optic neuritis. In this stage the mucous membrane of the middle ear becomes inflamed, especially that part lining the tympanic cavity; as a result there is an exudation of inflammatory products consisting of pus, blood, &c.

The membrana tympani almost always becomes softened and ulcerated, or thinned and weakened from pressure, and perforation follows.

The appearances of the membrane, as seen through the speculum in the early stage before perforation, are those of hyperæmia of varying intensity, the injection of the cutaneous vessels being most marked at the periphery of the membrane, the pars flaccida, and the handle of the malleus; in some cases of more acute nature the whole of the drum becomes of a bright red colour.

Later, as the inflammation increases, exudation occurs and pus is secreted. There is now seen to be a general or localised bulging of the swollen membrane, such saccular bulgings being usually of a yellowish green or red colour. Purulent exudation may, however, be present in the tympanic cavity without any bulging of the membrane, the latter having been altered and thickened by previous disease.

The superficial structures over the mastoid may be swollen, and the glands below and behind the ear enlarged and tender.

The duration of the first stage is variable; rupture as a rule takes place in from three to five days, and is commonly followed by cessation of the pain, and more or less profuse discharge.

Treatment.—The following are the lines of treatment, modified, of course, according to the severity of the symptoms:—The patient should be kept quiet both mentally and physically, given a good saline purge and a light diet. If there be severe pain, febrile symptoms, and considerable hyper-

æmia, the patient being an healthy adult, the local abstraction of blood by means of leeches (eight to twelve for an adult, two to four for a child) applied to the mastoid region, or tragus, and followed by the application of warm fomentations will give relief.

In less severe cases, or in people with broken-down health, where bleeding is contra-indicated, warm sedative poultices may be applied externally, or medicated applications be instilled into the ear every three or four hours. Of such applications solutions of opium and belladonna, of morphine (two grains to the ounce), of simple tincture of opium are the most useful. A few drops, eight to ten of the particular solution selected are warmed and dropped into the ear, and the canal closed with a pad of cotton wool.

In private practice a convenient form of medication consists of aural ovoids. They are capsules of gelatin containing one sixth grain of liquid extract of opium, or one twelfth grain of hydrochlorate of morphia, and are introduced deeply into the auditory canal with aural forceps, the meatus being then closed with cotton wool. The capsule gradually dissolves and the pain is relieved. Should the pain be so severe as to prevent sleep, ordinary internal narcotics are indicated.

When the pain, notwithstanding this treatment, continues, and there is evidence of a collection of pus in the middle ear, as revealed by yellowish bulging of the membrane, it is necessary to take measures to evacuate the pus in accordance with ordinary surgical principles.

It is of especial importance not to delay, for the tympanic cavity is in anatomical relationship with most important parts, and it is no exaggeration to say that a timely incision may be the means of saving the life of the patient. Moreover, paracentesis or incision with a myringotome not only gives exit to the pus in the tympanic cavity, but by division of the vessels reduces the hyperæmia and lessens the amount of damage which usually occurs in the membrane from spontaneous perforation; this is particularly the case when scarlet fever is the cause of the inflammation.

The operation is most conveniently performed as follows: The external auditory meatus having been thoroughly cleansed and dried, a 15 or 20 per cent. solution of cocaine is sprayed on to the tympanic membrane, a suitable speculum inserted,

and a good light reflected down the canal. If from the swelling of the external meatus the membrana tympani cannot be seen, it is wiser to stick to fomentations, &c.

The patient's head being held by an assistant, the membrane is freely incised from above downwards with a myringotome in the situation where there is the greatest bulging, or, if general, behind and below the handle of the malleus.

It is of importance that the incision should be a free one, as the contained pus is often thick and viscid, and moreover there is a tendency for the artificial perforation to close up too soon. After incising the membrane the middle ear should be inflated with Politzer's bag or a Siegle's pneumatic speculum, used in order to drive out as much pus as possible.

The auditory canal is then closed with aseptic cotton wool; the incision must be kept patent so long as there is any discharge of pus. The patency of the artificial opening is best assured by free incision and daily Politzerisation: but should there be a tendency for it to close too soon, a probe should be frequently passed through the aperture. Antiseptic cleansing should be carried out twice a day, and a lotion prescribed for syringing the naso-pharynx, otherwise the condition is apt to become chronic.

Illustrative case.—A. L—, æt. 13, came to my out-patient's department at Westminster with a history of severe pain in the right ear, radiating to the throat and head, and of three days' duration. He had been deaf for some six months, but beyond this had had no ear trouble; the pain was so severe as to prevent sleep and to cause him to cry out occasionally.

On examination of the membrana tympani, considerable peripheral hyperæmia was visible, and there was a yellowish bulging in the posterior inferior quadrant of the membrane. The boy had adenoids and naso-pharyngeal catarrh. The bulging was freely incised from above downwards under cocaine, and thick muco-pus evacuated. With Politzer's bag some more pus was driven out; the canal was then cleansed, plugged with salicylic wool, and a saline purge ordered.

He was instructed to gently syringe the ear twice a day with boric lotion. In eight days the pain and discharge of pus had gone, and the incision was healing.

He had no further trouble, and some weeks later I removed the adenoids. Six months after he remained quite well, the scar of the artificial opening was invisible, he could breathe normally, and his hearing was almost normal.

2. The Stage after Perforation.

(a) *Stage of purulent discharge.*—In this stage there is a more or less profuse discharge of pus from the external auditory meatus, and on examination of the membrane a perforation is seen. Usually, though not always, when perforation takes place there is a marked diminution of the severe pain and acute symptoms of the first stage. The nature of the discharge varies greatly; it may be thin and watery or thick and tenacious; there may be an excess of mucus, muco-pus, or blood may be intimately mixed with the purulent secretion. (In some cases the discharge is almost pure blood—otitis media hæmorrhagica.)

The discharge often smells horribly, having a peculiar sickly odour; this is very common in old-standing and neglected cases, and is sometimes indicative of carious or necrosed bone. A microscopic examination of the secretion may reveal bone *débris*. In other cases the pus tends to form, with the desquamating epithelium, dry masses (cholesteatomata).

From the constant irritation of the purulent secretion the external meatus may be found inflamed, its lining membrane being swollen and reddened, and sometimes ulcerated; in many cases a concomitant pustular eczema in the neighbourhood of the meatus results.

Deafness of variable degree, tinnitus, vertigo, occasional attacks of pain (usually due to some complication), enlarged glands, general anæmia, and occasional albuminoid degeneration of the internal organs, are among the symptoms that may accompany a chronic otorrhœa.

The duration of the second stage varies within the widest limits. Some cases heal up within a few days, others within a few months, many after several years, and some, again, continue for a lifetime. It is especially among the poorer classes that the worst cases are seen, or among the better class, where no particular notice has been paid to the discharge. The discharge may be constant or intermittent for a few weeks or months.

It is during this stage that the numerous serious complications of otorrhœa may arise, and I cannot too strongly impress on the minds of medical practitioners the fact that every patient who is suffering from a perforation and discharge from the ear has a veritable sword of Damocles hanging over him. At any moment one of the terrible complications of otorrhœa may arise and put an end to his existence.

The more I see of these cases the more convinced do I become of the supreme importance of urging on the patient and his friends the absolute necessity of carrying out the treatment ordered, in view of the grave dangers to which they are subject if left alone.

The fact of a patient having had a chronic discharge for perhaps many years, and no serious complications arising therefrom, is no guarantee whatever of his future immunity.

It is especially in the chronic purulent stage these dangerous complications are likely to arise. In the acute stage, extension of the pathogenic processes to the brain or its coverings is rare; this is probably due to the fact that the bone and mucous membrane of the middle ear are still intact, and these offer an obstacle to the invasion of the deeper tissues by the micro-organisms.

In the chronic stage the progress of the disease is insidious and often painless, but nevertheless progressive. Extensive destruction of the middle ear and its surrounding cavities occurs, and the meninges, blood-vessels, and brain are all liable to become attacked. If properly treated the purulent process may end in healing by cicatrization, the most favorable termination; or the purulent process having ceased the perforation remains open, a dry perforation resulting.

(b) *The non-secreting stage.*—With a permanent dry perforation a patient is always liable to an acute attack of inflammation by the loss of the protective membrane, and the consequent exposure of the tympanic mucosa to outside influences, such as cold, &c.

The diagnosis of a perforation is in the majority of cases easy. In any given case of otorrhœa, before attempting to get a view of the membrane, it is necessary to cleanse the auditory canal. This may be done either by careful syringing with warm water or weak boric lotion, and then drying the parts with pieces of absorbent wool twisted on an

aural probe, or, if the syringing causes giddiness and a tendency to fainting, by cleansing the canal with dry wool alone.

In very nervous patients the aural speculum should be warmed before insertion, and a little cocaine solution (10 per cent.) be sprayed into the meatus.

If the perforation be small or situated in the lower and front part of the membrane, and there be bulging of the anterior inferior wall of the external auditory meatus, great difficulty may be experienced in determining the perforation by inspection alone. I have also seen students mistake the reddened inner wall of the tympanum for an inflamed membrane, and *vice versa* in those cases where the whole of the membrane is destroyed.

Confirmatory evidence can be got by using the diagnostic tube and inflating with a Politzer's bag, by Valsalva's method, or the Eustachian catheter; the air will usually issue with an audible whistle or rushing sound.

A patient will sometimes tell you he tastes the purulent secretion in his mouth, and if fluids are poured into the ear they may pass down into the throat through the Eustachian tube.

Bubbles of air may also be seen if fluids are poured into the ear and inflation practised. In many cases Siegle's pneumatic speculum is of great use, especially when adhesions exist between the membrane and tympanum.

When inspecting the membrane for a perforation, note will be made of the absence or presence of granulation tissue or polypi; of any caries or necrosis of the ossicles or walls of the tympanum, and of the condition of the mastoid.

Treatment.—In the early stages just subsequent to perforation, I order the following treatment to be carried out: The ear to be carefully and gently syringed three or four times a day with a warm solution of boric acid (8 grains to the ounce). Whilst part of the solution is still in the meatus inflation by Valsalva's or Politzer's method is employed; this forces some of the intra-tympanic secretion out into the meatus, from whence it can be removed by again syringing out. The ear is then carefully dried and gently plugged with absorbent wool.

If the discharge be slight in amount syringing should be employed less frequently, and after drying the ear small quantities of finely powdered

boric acid are insufflated. Under this treatment recent cases will get well in a few days to a month. It is generally necessary that the practitioner should carry out the treatment himself, or else thoroughly instruct the patient's friends in the act of syringing and cleansing, so that it shall be properly done.

Among my notes I find records of several such cases, cicatrization having taken place within a month or less. Perforations due to trauma and followed by suppuration, as a rule, do particularly well under the above treatment. I have had recently two such cases; in one, a lad aged 18, the drum was perforated by the spoke of a bicycle wheel and suppuration ensued; in the other, a lunatic girl, the perforative otorrhœa was caused by a rusty nail secreted in the meatus; both cases were cured within three weeks.

Chronic cases which, unfortunately, are by far the most common, are often extremely difficult and even impossible to cure.

The general lines of treatment should be—

(a) Thorough cleansing and disinfection of the cavities of the middle ear.

(b) Appropriate treatment of the nose, nasopharynx, and throat.

(c) Careful general treatment especially with regard to such diatheses as tubercle, syphilis, &c.

Thorough cleansing and disinfection of the cavities of the middle ear are of the greatest importance, and such treatment is only in accordance with ordinary surgical principles.

Two methods may be employed—the moist and the dry. The former is applicable when there is profuse and foetid discharge; the latter when syringing causes pain or giddiness, where the discharge is slight and non-foetid, and in cases of old quiescent perforations (if they are treated at all). I carry out the moist treatment thus: Thorough syringing of the ear with warm boric lotion (100°—102°F.); inflation of the middle ear by Valsalva's or Politzer's method, and careful drying of the parts with absorbent wool wicks twisted on an aural probe; finally, I insufflate a very little finely-powdered boric acid on to the diseased mucous membrane of the tympanum, and lightly fill the outer half of the external meatus with a plug of salicylic wool. If the discharge be very profuse I order this treatment to be carried out three or four times a day. As the discharge

lessens, the syringing, inflation, drying, and insufflation are less frequently done, until finally the powder remains dry for days or even weeks.

This treatment may have to be persevered in for a considerable length of time before a permanent cure results; if no benefit accrues after a thorough trial, other methods or medicaments must be employed.

Of course, such complications as granulations, polypi, caries, &c., will require special attention before the boric acid treatment can be expected to be efficacious. The next most useful solution in my experience is peroxide of hydrogen (fifteen to twenty volumes strength); this solution is of particular value in tubercular cases.

Of other solutions for syringing, a choice may be made from the following: carbolic acid in water (2 per cent.); corrosive sublimate solution (1 in 3000—4000); a solution of boric acid in water and alcohol (boric acid 1, distilled water 30, rectified spirits 5·30); a teaspoonful of 10 per cent. alcoholic solution of salicylic acid to a wine glassful of water; glycerin of carbolic acid and liq. plumbi diacetatis (1 ounce to 10 ounces of water) a dessertspoonful in a wine-glassful of warm water).

In the employment of these different solutions a choice must be made according to their antiseptic properties or any injurious micro-organisms present, and with reference to the nature of the secretion itself, whether thick and tenacious or thin and watery, and according to the presence or absence of foetor.

Treatment by rectified spirit and strong astringents. The use of rectified spirit, first introduced by Weber-Liel, is a method of treatment often of as much service as the boric acid. It is especially indicated when there is much granulation tissue present, and after polypi have been removed.

After cleansing and drying the ear, the head is inclined well over to the opposite side, and from ten to fifteen drops of the warmed solution (usually, to commence with, equal parts of spirit and water), instilled into the ear. The drops are kept in for ten minutes; the solution is then allowed to flow out, the parts are dried, and the canal plugged lightly with salicylic wool. This treatment is repeated two or three times a day, and the strength of the spirit gradually increased until it can be borne pure.

I frequently use the spirit drops in combination with a solution of boric acid in water (*vide* solutions), and find it often more efficacious than either alone.

If there be much foetor it is well to add a little carbolic (2 per cent.) to the solution. Iodoform may be combined with the alcohol, half a drachm of the powder to an ounce of the spirit.

In very obstinate cases, with extensive destruction of the membrane, excessive granulation tissue, and great swelling of the mucous lining of the middle ear, astringents or caustic applications are indicated. Ten to fifteen drops of a 10 per cent. solution of nitrate of silver are poured into the ear in the same way as the spirit drops, and when allowed to run out again the ear is syringed with a solution of chloride of sodium to neutralise any of the silver salt remaining; or the diseased mucous membrane may be rubbed over with a solution of the strength of 40 grs. to the ounce.

In a few cases, where there have been no signs of any acute inflammatory mischief, I have carefully touched the diseased mucous membrane or granulations with chromic acid fused on a probe, and have obtained good results. It must be very carefully applied as the caustic action of the chromic acid is very great.

The dry method.—The lessening of the discharge is an indication to diminish the frequency of the syringing, and to employ dry applications. The ear is cleansed with cotton wool-wicks and the diseased parts are insufflated with finely-powdered boric acid. A powder formed of boric acid four parts and iodoform one part may be substituted for the plain boric acid, particularly in tubercular cases.

(b) *Appropriate treatment of the nose, naso-pharynx, and throat.*—In every case of otorrhœa it is incumbent on the practitioner to make a thorough examination of these regions. As I have already stated, the majority of cases of perforative otorrhœa originate in some morbid condition present in the naso-pharynx, &c., and it is as necessary to treat these morbid conditions as to treat the middle ear itself. Enlarged tonsils, adenoids, hypertrophic rhinitis, spurs, nasal polypi, nasal or post-nasal catarrh, must all receive the special treatment appropriate thereto.

It is unwise to remove adenoids or tonsils if there be profuse *fœtid* otorrhœa. An attempt

must therefore be made to render the middle ear as antiseptic as possible, and then the particular operation may with safety be undertaken. For nasal or naso-pharyngeal catarrh I order daily syringing of both nostrils with one of the two following solutions :

Sod. Chlorid.	}	... āā grs. vij
Sod. Bicarb.		
Boracis		
Sacch. Alb.	...	grs. xv

One powder to be dissolved in half a tumblerful of warm water and used with the syringe. Or

Glycerin. Acid. Carbolic	...	℥xij
Sod. Bicarb.	...	grs. vj
Boracis	...	grs. vj
Aq. destill. ad	...	3j

(c) *General constitutional treatment.*—If syphilis or tubercle be present they must receive appropriate treatment. Good food, fresh dry air (mountain air), good sanitary surroundings, and tonics, especially iron, must be recommended.

The patient must be warned against exposing himself to cold draughts, and told to carefully avoid the entry of cold water, especially sea water, into the ear or ears.

It is only by careful attention to all these details that success to any degree is likely to be obtained. The patient or his friends must be told of the possible consequences of the disease ; and strongly urged to persevere in the plan of treatment laid down by the surgeon, although it may be a long while before healing takes place.

Illustrative cases.—CASE 1. W. P—, male æt. 4, attended my out-patients February, 1896. For two years constant profuse discharge from left ear following measles. Had treatment at home from a medical man. Fair-sized central perforation noted. No caries or polypi. Watch on contact only.

Ordered daily syringing with Lot. Ac. Boric., Politzerisation, drying, and insufflation. He was also ordered Coll. Sodii for nasal discharge.

By middle of May dry treatment commenced.

By September he was cured, and his hearing had greatly improved.

January, 1898.—Quite well, cicatrix, hearing good. Wears a wool plug.

CASE 2. Fred. R—, æt. 5½. Measles twice, last time in June, 1895, since then double perforative otorrhœa. Discharge thick, constant, profuse, and

foetid. Attended out-patients August 2nd, 1896. The same treatment was carried out as in Case 1.

By August 17th, discharge from left ear ceased. September 14th.—Hearing much improved.

October 24th.—Discharge from right ear ceased. 29th.—Severe pain of left ear, discharge, and relief.

December 3rd.—No discharge from either ear.

January, 1898.—No discharge from either ear since November, 1896. Hears well.

CASE 3. Rosina R—, æt. 9. Post-scarlatinal discharge left ear for over two years. Been attending hospital all the time. Came under me September 1st, 1896. Large perforation, left membrana tympani noted. Mucous membrane of tympanum swollen and granular. Treated with spirit drops and Lot. Ac. Borici.

October 26th.—Discharge had gone.

May, 1897, still remained well, dry perforation, wears a cotton wool tampon, and hears fairly well. Seen six months ago quite well.

These are three out of many similar cases, and show what systematic and persistent treatment can do.

There are certain cases in which the perforation is of such a nature or in such a position as to require special treatment.

Perforation in the membrana flaccida or Shrapnell's membrane is, in my experience, a condition very difficult to cure. The pus located in the middle ear is unable to escape freely, and attacks suggestive of brain trouble and due to retention are common. Moreover, perforation in this neighbourhood is often associated with caries of the attic and antrum, and serious complications are very apt to occur. I always, therefore, look upon such cases as of grave import. In such cases some surgeons enlarge the opening by careful sharp spooning of the free edge of the membrana tympani, and subsequent syringing out with the intra-tympanic syringe and instillation of spirit drops.

I have had good results by this method in two patients.

Poltizer removes the outer wall of the attic with special forceps ; Schwartze with a fine chisel and hammer. I have not employed these means. If enlarging the opening by scraping the free edge of the membrane and subsequent intra-tympanic syringing fails, I open up the mastoid antrum.

A small perforation with thick secretion presents the same difficulties, namely, impeded exit of the secretion, and prevention of proper cleansing of the middle ear. Enlargement of the perforation, in my opinion, is the proper mode of dealing with the condition.

In this short paper I do not intend to deal with the consequences that may arise from a neglected otorrhœa, it is far too extensive a subject; yet as I wish to lay stress on the especial operative treatment that should, in my opinion, be recommended in those cases of intractable purulent otorrhœas which have resisted all ordinary forms of treatment, and as the recommendation of these operative measures depends on the possible super-vention of these dangerous complications it is as well to enumerate them. The complications that may arise are as follows: aural polypi, acute mastoid periostitis, acute or chronic collections of pus in the mastoid cells, antrum, and attic, facial paralysis, meningitis, cerebral and cerebellar abscess, purulent thrombosis of the lateral sinus and internal jugular vein, with embolic pneumonia and general pyæmia, lardaceous disease, general tubercular infection, glandular abscesses, rarely profuse hæmorrhage, and still more rarely epithelioma of the tympanum or external auditory meatus.

This list of complications pretty well indicates the significance of neglected otorrhœa.

In my aural practice I get a very large number of patients, especially hospital patients, who have been suffering from otorrhœa of middle ear origin of several years' duration; some of these have undergone an extensive course of routine treatment; others, on the contrary, have neglected treatment altogether. In the absence of any complication or special peculiarity, I try the usual routine treatment carefully, and for some length of time; if no improvement accrues after this lengthy and varied treatment I strongly advocate mastoid antrectomy or attico-antrectomy.

This I look upon as the *radical cure* for chronic otorrhœa, and having within the last three years performed the operation twenty-six times or more, and with extremely satisfactory results, I can, with the confidence born of experience, thoroughly recommend the operation in suitable cases.

As I have already stated in this paper, chronic otorrhœa is too often looked upon as a condition of no particular importance, instead of a menace to

life, and under these circumstances it is the duty of the medical attendant not to rest satisfied until the discharge is permanently cured. Macewen ('Pyogenic Diseases of the Brain and Spinal Cord,' p. 293), says, "Where the tympanic cavity has become the seat of chronic suppuration, with ulceration of the mucous membrane extending into the antrum and mastoid cells, it becomes a standing menace to the safety of the patient. The disease progresses insidiously, and one cannot be certain as to where and when it may end. A person might as well have a charge of dynamite in the mastoid antrum and cells, as one cannot know the moment when accidental circumstances may arise which may cause the infective matter to become widely disseminated all over the cerebro-spinal system."

The objects obtained by the operation are:

(1) The removal of granulation tissue, diseased mucous membrane, cholesteatomatous masses, and carious or necrosed bone contained in the antrum and attic.

(2) Free drainage of the middle ear and antrum.

(3) The prevention of extension of the inflammatory process to the brain and meninges.

The radical operation.—I employ, according to circumstances, one of two methods of operation, namely (1) *Antrectomy* or ablation of the mastoid antrum and cells, with removal of infective material from the middle ear, and with or without extraction of the ossicles through the same opening. (2) *Attico-antrectomy*, or the same steps as in antrectomy, with the additional removal of part of the posterior superior wall of the osseous meatus and opening up of the attic, thus throwing the antrum, attic, middle ear, and external meatus into one large cavity.

Among those surgeons who have had most experience in these operations, attico-antrectomy is most generally practised, and seems to give the best results and the greatest immunity from relapse.

Attico-antrectomy, however, is in my experience a much more difficult and serious operation to perform, and requires considerable prior study on the cadaver. At the same time I quite agree that it gives more consistently satisfactory results than antrectomy alone. I find on referring to my notes, and taking my first twenty-six cases, that in nineteen I performed antrectomy, and in seven attico-antrectomy.

The results were as follows :

Antrectomies.—19 cases.

Cured	14
Not cured	5
			—
			19

Of the five cases not cured three were much benefited, the discharge being slight and odourless. In two failure resulted.

Attico-antrectomies.—7 cases.

Cured	6
Failure	1
			—
			7

The one failure was in the same patient in which a failure was registered among the antrectomies. The patient not having benefited at all from the opening up of the mastoid antrum, I later performed attico-antrectomy, but unfortunately without success, and, moreover, with the result that facial paralysis supervened.

As I intend later to make these operations the subject of a special paper, and to describe the operations, their indications, difficulties, and dangers, I will only reiterate what I have said before, that owing to the great advance made in the operative treatment of these neglected cases of otorrhœa, which are not amenable to ordinary and persistent routine treatment, and which are so frequently followed by serious complications, the practitioner can with confidence advise what I have, in common with others, called the radical cure.

The Dangers of Typhoidal Urine.—Dr Petruschky ('Centbl. f. Bakt.,' xxiii, No. 14; Ref. 'Med. Age,' xvi, No. 12, p. 379).

The author gives the results of looking for typhoid bacilli in the urine of fifty typhoid patients. In only three cases were they found in the urine, and in only one case could they easily be detected in the fæces. Although the fact that typhoid bacilli are eliminated with the urine has been known for some time, they are seldom present, and not in the beginning of the disease, so have no diagnostic value. Petruschky, however, believes that they are a great factor in the epidemics of the disease.

The Post-Graduate, September, 1898.

RIGORS: THEIR CLINICAL SIGNIFICANCE, AND THE CONDITIONS UNDER WHICH THEY OCCUR.*

BY

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MR. PRESIDENT, LADIES AND GENTLEMEN.—In selecting a subject for a short paper to read before you this afternoon I was at first at a loss to know what to choose. I knew that the time allotted to me would be limited, and I knew also that I had no original matter to offer; and when I reflected upon the vast amount of literature, in the shape of text-books, systems, and journals, that are daily being published for our edification, the possibility of my being able to add to your knowledge (for I take it we all expect to glean a little information from these papers) seemed hopeless. However, I finally decided not to aspire too high, and to content myself with the study of a single symptom only, and to look at it, not from the point of view of any one particular disease, but in its relation to some of the various conditions under which it occurs, and I have taken the subject of rigors because it has been my good fortune of late to have met with this symptom under conditions where it is not ordinarily described as occurring, for which reason I must conclude it is unusual.

I will preface my remarks by saying that rigors may be divided into (1) true and (2) false or spurious rigors, the former being the well-known shivering fit, with which you are all familiar; the latter being a condition which has the outward manifestations of the former, though in a less marked degree, and in which there is no rise of temperature.

To-day we are concerned with the true rigor only, but to give you an example of what I mean by a false rigor it is unnecessary for me to go outside our ordinary daily experience, I need only mention the momentary sensation of a chill and

* Read at the Autumnal Meeting of the East Anglian Branch of the British Medical Association, held at Mundesley-on-Sea, September 8th, 1898.

shivering we feel immediately after passing water, which I take to be an apology for the more serious and pronounced condition which is so often the sequel to the introduction of a catheter. Other examples of false rigors will doubtless suggest themselves to some of you.

Knowing as we do so little as regards the pathology of rigors, it is difficult to give an accurate classification of them, for I think there can be no doubt that the causative agency varies under different conditions. However, for purposes of convenience, I have divided them into rigors of toxic origin, and rigors of nervous or other obscure origin; though I must admit it is difficult sometimes to know into which category one should place the case before one.

Toxic rigors.—Perhaps the most familiar example of these are the rigors occurring in pyæmia. In this disease we may have them recurring daily, or at longer or shorter intervals. I have notes of one fatal case following pregnancy, whose chart I will pass round, where at least one severe rigor occurred every day for thirty-one days; and there has recently been in the hospital a case of tubercular disease of the bladder, with pyæmia, in a boy aged eleven, who on one occasion had three bad rigors in the space of sixteen hours. And I may here remark that I know of no disease, excepting pyæmia, where one meets with rigors occurring more than once in the twenty-four hours.

In pyæmia, as is well known, the poison is circulating in the blood, though it does not multiply in it, and there is undoubtedly a relation between the rigors and the formation of metastatic abscesses, though how close this connection is it is difficult to say.

A rigor marks the onset of many acute septic conditions which cannot be classified as cases of pyæmia. Of 100 cases of acute peritonitis recorded by Mr. Treves,* rigors were noted in 13 cases, and of these 10 died.

In an acute uncomplicated case of abscess of the brain, whether secondary to otitis media, traumatism, or other source of infection, a rigor is a constant early symptom. Repeated rigors in a case of intra-cranial mischief, secondary to middle ear disease, are very suggestive of a disintegrating infective clot in the lateral sinus, and this suspicion

is confirmed if there is evidence of infective infarction in the lungs or elsewhere.

In no department of surgery, I think, is there a better illustration of treatment, being based upon an exact knowledge of pathology, than is exemplified by the modern treatment of these cases of lateral sinus pyæmia. But this by the way.

In septic cerebro-spinal meningitis rigors are common, and their presence are an aid in diagnosing this condition from meningitis of tubercular origin.

Akin to ordinary surgical pyæmia is another disease in which rigors not unfrequently occur, and when they do occur are of great diagnostic importance. I refer to infective, or as it is commonly, though less accurately termed, ulcerative endocarditis, which might quite well be called arterial pyæmia. Now this is a disease which, perhaps of all others, is most likely to baffle the skill of the practitioner, and yet how important it is to be able to recognise it, especially when one remembers the very high mortality that attends it. Into the question of its differential diagnosis I will not enter now, but will only say that should the disease be suspected, and rigors occur, then the suspicion will have been well founded; and further, should symptoms of embolism occur later then the diagnosis will be made certain.

In certain forms of phthisis, where there are multiple abscesses in the lungs, and fresh ones being constantly formed, in which both staphylococci and streptococci are found, and which are always associated with high fever, we should almost expect to meet with rigors, and the wonder is, I think, that they do not occur more often. But that the symptom is present in some of these acute cases there is no doubt. I personally can only call to mind two cases in which I have met with them, but then one's own experience is often so very misleading. Perhaps some of those present who have had more opportunities than myself of watching such cases can give us further evidence on this point.

In the acute disseminated form of pulmonary tuberculosis rigors are more common; and Dr. Kingston Fowler remarks, in his recently published book on diseases of the lungs, that there may be repeated rigors in this condition.

I have only once met with rigors occurring in an uncomplicated case of jaundice. I put aside, of

* Allbutt's 'System of Medicine,' vol. iii, p. 614.

course, the cases of catarrhal jaundice in which there is often a sensation of chilliness during the first day or so of the illness. In this case, that of a man aged fifty-nine, the jaundice was of the obstructive variety, and had existed for about twelve months. It was caused by a non-malignant stricture in the common bile duct, as was proved post-mortem. I quote the case as another example of toxic rigors, for they were evidently due in this case to the cholæmia from which the man was suffering; and it also impresses upon us the fact that, although we usually associate rigors in jaundice with some suppurative condition going on in the liver or gall-bladder, yet this is not invariably so.

Of the many conditions in which rigors occur it is in malaria, perhaps, that they can best be studied, for in this disease we can often foretell, within an hour or so, when the shivering fit will commence; and in ague it is not uncommon for the whole cycle of events to be spread over some hours of time.

The accompanying chart well illustrates a case of tertian ague I once had under my care. There was no doubt about the diagnosis, because on several successive occasions I found the malarial parasites in the blood. In this case I thought I would see what effect a drug like pilocarpine would have upon the attack. To this end I gave hypodermically $\frac{1}{2}$ grain of the nitrate at the very commencement of a shivering fit, and had the rectal temperature recorded every half hour for the next six hours. The dose was a full one I admit, and the effect produced was comparable. The patient went on shivering, but instead of his temperature rising as it ought to have done, it rapidly sank until it had got below 97° F., and during this time he was sweating profusely, and liquid was running from his eyes, nose, and mouth. After about two hours the shivering and sweating gave way to a sensation of warmth, and at the same time his temperature rose a little and eventually reached 100° F., whereas, in previous attacks, as you see, it had been mounting up to 104° F. and 105° F. This patient was kept under observation for a further period of twelve days without his having another attack.

I have never before nor since heard of pilocarpine curing ague, and if any members present should ever be tempted to try the drug for that

purpose, I should strongly recommend them to take this warning from one who, in the ardour of his youth, gave an heroic dose of a very powerful drug, which certainly had the effect of aborting the rigor, but which at the same time very nearly killed the patient.

With regard to the occurrence of rigors in typhoid fever I will say little, since most of you have had considerably more experience of this disease than I have, and have doubtless noticed their presence. I have seen an initial rigor the earliest symptom, which greatly increased the difficulty in diagnosis, and I have met with rigors occurring during the course of the illness. In this disease it is a symptom, I think, which alarms more than it harms, and I should not say the prognosis is much affected by its occurrence, unless of course it is frequently repeated, or is the manifestation of some septic or other complication.

That many of the so-called acute specific diseases—such as pneumonia, erysipelas, influenza, &c.—are ushered in by a rigor is, of course, common knowledge, and this mode of onset will often help us, by the method of exclusion, in arriving at a diagnosis. That these rigors are of toxic origin there can, I think, be little doubt. Most, if not all, of these diseases can, I think, even now be said to have a microbic origin; and if we take as an example influenza, in which the patient is often without any warning suddenly rendered utterly prostrate, we can only suppose that such a condition of collapse is brought about by a large dose of the influenza poison circulating in his system, and it is this poison, I imagine, which is the cause of the rigor.

In influenza definite rigors have been described by some as of daily occurrence in certain forms of the disease, but I have not met with such cases; and where they recur with such regularity there might be difficulty in diagnosing the condition from pyæmia and ague.

Most primary anæmias, such as leucocythæmia, pernicious anæmia, &c., are accompanied by a variable amount of fever of an irregular type, but I can find no reference to rigors occurring in these conditions. I have seen rigors occurring daily for some weeks in a case of splenic leukæmia in a girl aged 18, and I have also met with rigors as a symptom in lymphadenoma, which, although not an anæmia of the same pernicious type as that I have just men-

tioned, must nevertheless, at any rate for the present, I think, be placed in the same category. You may say "but these can scarcely be called toxic rigors," and that may be so; yet many are of opinion that these diseases are of bacillary origin; and, if not, that they are, as I think more probable, due to an as yet undiscovered hæmatozoon, similar to that, perhaps, which is now known to be the cause of malaria.

I will only mention one other class of toxic rigors before giving you an example or two of those of more obscure origin.

In food poisoning, whether resulting from the ingestion of tinned food, ice-cream, or other articles of diet, the symptoms, of which a rigor may be one of the earliest, are often so severe as to lead to a fatal issue. The toxicity here depends upon two causes, viz. the bacterium, and the chemical products of its activity. If due to the former there will be an incubation period of variable duration, during which the micro-organisms are manufacturing the chemical poisons, or ptomaines, which produce the rigor and other symptoms; whereas if symptoms show themselves within a few hours of eating the suspected food, then they are due to ptomaines existing as such in the food before it was taken.

And now a word or two upon rigors of nervous or other obscure origin.

The most typical example of a nervous rigor is that which follows the introduction of a catheter. That the rigor which so often results from this apparently trivial operation is of a reflex character there can, I think, be no doubt, though why it should sometimes occur almost immediately afterwards, and at other times not till many hours afterwards, is almost as difficult to explain, to my mind, as it is impossible to foretell whether or not a rigor will occur at all. These cases, however, rarely come under my observation, and perhaps the surgeons can better explain them.

During the last year or so the transfusion of a solution of common salt (3j—Oj) has been resorted to in several cases in the Norwich Hospital to overcome the shock attendant upon injury, operation, or other cause, and by its means the lives of several patients have been saved. The solution—one to four pints at a time—has been given intra-venously, and brandy, to the amount of 3ss—Oj, has sometimes been added.

The results of this treatment have been very striking, but the only after-effect which concerns us now is the rigor which so often occurs within an hour or so of the injection. It does not always occur, and it is impossible to say when it may be expected. In some cases, where the transfusion has been repeated two or three times at intervals of only a few hours, a rigor has been noticed after the first injection only; and in a case of diabetic coma, where it was done to restore consciousness, five injections, each of about three pints, were given in the space of seventy-two hours, and there was a definite shivering fit, without any rise of temperature, after the first injection, though no such phenomenon was observed after any of the subsequent injections. The first case of this kind that I saw was in a woman aged 30, who had undergone a very extensive operation for glands in the neck, which was followed by a great deal of subsequent shock, for which she was transfused with three pints of saline and brandy. One hour afterwards she had a severe rigor lasting half an hour, which both Mr. Ballance and I (for we were both resident in the hospital at the time) took to be the initial rigor of acute septicæmia. She was accordingly given large doses of antistreptococcic serum, and she got perfectly well. But I have now no doubt, in the light of subsequent experience, that this patient would have got well just as quickly had she not been subjected to this serum treatment. It is rather difficult to explain these rigors, though they are probably due to a large amount of fluid being sent to a previously very anæmic brain. That it is not the salt or the brandy which produces them is proved by the case of diabetic coma above referred to, in which the brandy was omitted, and sodium bicarbonate replaced the ordinary sodium chloride.

In connection with intra-venous injections I will mention the sudden and often rapid rise of temperature, reaching it may be 103° F. or 104° F., sometimes associated with a rash, at other times not, which, doubtless, many of you have noticed to follow the subcutaneous injections of the various antitoxins. A slight rigor sometimes accompanies this temperature. This disturbance can hardly be due to the antitoxin because it is not seen at all when using the serum of certain horses, while with that taken from others it is frequent. I have not noticed this after-disturbance so much of late, since

the antitoxins have been sent out more concentrated, which certainly suggests that it is due to some normal or abnormal constituent in the serum other than the antitoxin.

It is unusual to meet with rigors in children, but we have lately had in hospital a child aged 3 years 10 months, with suppuration in the neck, who had three well-marked rigors during his illness. I have not met with the symptom at an earlier age than this.

I have never seen or heard of a patient dying in a rigor, though when one witnesses a severe rigor one cannot help thinking that dissolution is imminent, and doubtless such would often take place if the disturbing influence lasted long enough.

The time at my disposal, gentlemen, has now come to an end, for there are other papers to follow, and I cannot better conclude than by thanking you for the kind attention you have given to the few remarks I have made in my attempt to handle in a concise, though by no means exhaustive, manner, a subject still wrapped up in so much instructiveness and obscurity.

Treatment of Exophthalmic Goitre.—Deguy says that the most important symptomatic signs to be combated are the cardiac disturbances which are usually manifested in symptoms of feebleness of this organ. In some of these cases digitalis is a valuable drug, in other cases it fails to do good. It is particularly indicated in those instances in which the heart is greatly disturbed in its rhythm and power. In these cases the digitalis should be given in small doses. The murmurs which are heard in the heart in many cases of exophthalmic goitre are not to be regarded as valvular affections, but as temporary symptoms of the disease. For the palpitations, cold compresses may be applied to the præcordium, or in their place atomisation by ether or chloride of ethyl, or the use of an ice-bag. In those instances where there is considerable cardiac pain or pseudo-angina, nitro-glycerine is useful. The best hypnotics for the insomnia are chloral and trional.

Monthly Cyclopædia.

CHAPTERS FROM THE TEACHING OF DR. G. V. POORE.

No. VIII.

GENTLEMEN,—Next, as to making post-mortem examinations of persons found dead. You should always carefully examine the clothing for stains, soiling, stabbing, &c., and notice whether stabs in the clothing correspond with stabs in the body.

I need hardly say that nothing requires more skill or practice than the making of post-mortem examinations. The experienced pathologist is the only person capable of properly interpreting the signs which are found after death. When you are called upon to make a post-mortem you must remember it is a very important duty, and one that has to be done with a great deal of care. Therefore, be sure that, when you start to make a post-mortem, you have plenty of time before you. You do not know what you are going to find; the cause of death may be something perfectly obvious, or, on the other hand, it may be something which is very difficult to find out, and you have got to continue with that examination until you are satisfied. Those who have had experience of this kind of thing must often have been aware of the fact that instead of half an hour or an hour they have been occupied five or six hours. In cases requiring judicial investigation, it is very important, seeing that the post-mortem examination must not be hurried, that it should be conducted in a proper place. In London there are now public mortuaries and coroners' post-mortem rooms, so that if an examination proves to be a long and difficult one it need not be finished at once, but the room can be locked up and things left, for the examiner to get a needed meal or rest. If you have to make a post-mortem examination in a remote place, you must take every single thing with you which is likely to be necessary. I had almost said you must take the water and towels and a bit of soap to wash yourself with. You can never rely upon getting anything. You must take not only what is mainly necessary, but trifles such as a piece of string to sew the body up again, scales to weigh the viscera with, which is a most important matter; a catheter to draw off the urine, a proper note-

book, and, in case it should be desirable to preserve any of the viscera, proper vessels, perfectly clean, to put them in. If you take these vessels you must also have the means of securing them, such as string and sealing wax and your own seal. I grant that you may not want some of these things in a hundred cases, but in the one hundred and first you may, and you may be seriously inconvenienced if you have not these appliances.

Always, if possible, take somebody with you ; do not try to make the examination single-handed ; take a medical friend with you who also has a fair knowledge of pathology. Then one can make the examination while the other writes the notes. Moreover, in that case the pathological appearances are submitted to two pair of eyes, and two judgments instead of one. But it is of the utmost importance that the notes should be taken. I think it is very desirable to take these notes of post-mortems in numbered paragraphs, and for this reason, that cross-references are thereby facilitated. The first thing to note in making a post-mortem examination are the signs of identity, therefore you should make a record of any scars. Some of you may have been present at the post-mortem yesterday afternoon on a body I had never seen before, that of a woman. Before the body was opened we were able to say that there was a scar on the left temple, that there was a big vaccination mark on the left arm ; we were able to say that one or two of the teeth were wanting ; we saw that the patient had two black eyes ante-mortem, and on making an incision into these we found distinct evidence of extravasation. We saw that there was œdema on the body, and that the body was fat, bloated, and so forth. There are certain other indications which lead to identity, such as tattoo marks and any deformity or abnormality of the body. Be sure to turn the body over, and look at the back as well as the front. Sometimes there are indications of occupation to be found about the body. I shall not now go into them in detail, but you can tell largely by the hands whether the deceased was a person habituated to hard manual labour, or whether the reverse was the case. One may sometimes get definite evidence of occupation. In works on 'Medical Jurisprudence' you will see many indications of occupation put down, and they are more or less true, but remember that hand crafts

are every day being supplanted by machine crafts, and the indications of occupation are probably less trustworthy than they were. Next note the state of nutrition. In a woman the presence of lineæ albicantes, a probable evidence of previous pregnancy, would be noted. You must carefully note the signs which indicate the time which has elapsed since death, such as the temperature, rigor mortis, and putrefaction, which I have been dealing with. In the next place, whether the body be that of a male or female, I strongly advise you to pass a catheter and draw off the urine. Only a short time ago a case was before the courts in which it was alleged that a servant girl had been starved to death. The prisoner was convicted, and got a term of penal servitude. Now it was said that servant girl was exceedingly thin, and that she had a voracious appetite and was very thirsty. The defence set up was a perfectly plausible one, and a very proper one, namely that the deceased might have suffered from diabetes, which would have accounted for the emaciation, for the appetite, and for the thirst. It was a perfectly legitimate defence. The question was one of great importance. Perhaps there was no urine to be got ; but if the urine had been examined, however little, there would have been the means of a complete answer. You have to remember that urinary diseases are a cause of death and of sudden death. Granular contracted kidney may give rise to coma and rather sudden death, and the examination of the urine will tell you whether it was albuminous, and whether it contained sugar. The establishment of either of those two facts would be of the greatest possible importance. You will appreciate how important it is to draw off the urine before making the examination on a body, because, if the urine is bloody and polluted, you cannot properly test for sugar or albumin. As we know very well, pathological conditions of the kidney are sometimes very difficult to appreciate, but the condition of the urine helps us in this direction. Then always examine a body carefully for signs of injuries externally. I would remind you that sometimes these external injuries are hardly noticed. For instance, a stab with a stiletto under a pendulous breast sometimes may pass unnoticed if it is not looked for, and there are cases where sharp instruments have been put up the nostrils. There is a record of a case in

which death resulted from the end of a clay pipe being driven up the nostril and through the ethmoid. That is a thing which requires to be hunted for, as it may easily escape notice.

Then, if there are wounds, note their position, their size, and their direction; pass a probe into them, and examine just as you would during life. To continue with the post-mortem, you have to remember that when you make a post-mortem for the coroner it is obligatory upon you to examine all three cavities of the body—the thorax, abdomen, and the head. That was brought to my attention when I was house surgeon. A man was admitted to the hospital smashed. He had fallen off a railway engine, and got between the engine and the wall. There were any number of injuries, any of which might have caused death; among other things he had a ruptured liver. The post-mortem was made, we saw ample causes of death, and there was an end of the matter until I was called one afternoon to give evidence to the coroner. Now comes the important point. There is no doubt the injuries I had described caused death, but how came the man to fall off the engine? The evidence went to show that one of his fellow workmen had negligently left one of those water pipes or hoses, such as are used to fill engines with, in a wrong position, and that the pipe caught this man as he was on his engine and knocked him off it. If that was the case it is possible that the man who had been guilty of this negligence had been guilty of manslaughter. The defence was that he fell off his engine in a fit. The coroner turned to me and asked, "What was the condition of the brain?" I had to say I had not opened it. It is a very good case in point. Therefore, you must always examine the three cavities, because you never know what the defence may be. Fortunately, in this case, the evidence of his ever having had a fit was practically *nil*, and the evidence of his having been knocked off the engine by the pipe was established by eye-witnesses, so that the question of a fit was not of very much importance here. But if there is a man being tried for his liberty or his life, defences of that kind are put forward, and the most will be made by the defence of any slip made by the doctor at the post-mortem examination. In making the post-mortem on the two rabbits the other day, we saw that the first appear-

ance on opening the abdomen was important. In the drowned rabbit you will remember the intestines were very dark coloured, showing much evidence of congestion. In the examination of the body yesterday afternoon the reverse was the case; the intestines were pale, and when I got the history, it showed that the patient died quite suddenly and unexpectedly. We found there was extreme fatty degeneration of the heart; the heart had stopped suddenly, and the circulation stopped before the respiration; therefore, the intestines were not engorged or congested. She did not die from asphyxia. An important point, and one which is insisted on by Virchow, is that in opening the thorax one should divide the costal cartilages right away down, and turn back the sternum and the cartilages, and then look at the pericardium and the pleura before dividing the sterno-clavicular articulation. The reason for that is that when you divide the sterno-clavicular articulation, you are almost sure to divide the great veins, and then you will get the thorax flooded with blood, and that will make you doubtful of the true condition of the pleura and pericardium.

I want to remind you again that the congestion of organs is more often passive than active, and depends upon the mode of death. Be very careful how you say that the parts are inflamed. It is very difficult to say that a part has been inflamed unless there be the products of inflammation, that is, unless there be exudation of lymph upon the surface, or pus. Again, I would say that where parts may have to be preserved as in cases of poisoning, you must be careful that your vessels are quite clean, and that after the viscera are put into the clean vessel they must be tied and sealed with your own seal.

I do not think you should let a stranger be at a post-mortem examination in doubtful cases unless you are satisfied that there are adequate reasons for his presence. I mention that because, in the noted case of the murder of Cook, by Palmer, the doctor of Rugeley, the murderer was present at the post-mortem. As the stomach was being opened Palmer jogged against the medical man who was collecting the contents of the stomach. It happens that they were not spilled, but the impact nearly knocked the medical man over, and there is no doubt it was intentional. The contents of the stomach were sent by postchaise to the

nearest railway station to be transmitted to Dr. Taylor, and it was proved that Palmer had offered the postboy £5 to upset the carriage. Therefore be careful who you have present at the inquest, and remember that a man who is likely to be tried on the capital charge, will not stick at trifles to escape the punishment which probably awaits him. Use your judgment.

Now, passing from generalities we come to particulars, and the first class of deaths which I take up are *deaths from poisoning*. First of all, we may ask what is a poison? Taylor's definition is "a poison is a substance which when absorbed into the blood is capable of seriously affecting health or destroying life." The law does not define a poison, and the words of the statute directed against the crime of poisoning are:—"Whoever shall administer or cause to be administered to or taken by any person any poison or other destructive thing with intent to commit murder, shall be guilty of felony." Therefore, if one administers, as has been done in India, powdered diamonds or glass, which is not a thing which will be absorbed into the blood, it would nevertheless be taken to be a "destructive thing." Then, "the administration of poison or noxious thing with intent to annoy or injure" is now a felony or misdemeanour, as the case may be. If a man insults you in the street and you knock him down, you may be punished, as you must not take the law into your own hands. And if, as sometimes has happened, servants have been suspected by their mistresses of purloining the whisky, and if into the whisky bottle is put a little tartar emetic, and if the person who drinks the whisky is made ill by that tartar emetic, she (or he) can be made answerable in a court of law. In this course I shall deal almost exclusively with poisons which have been used criminally. In these days we talk of toxins, which are poisons brewed in the body by pathogenic micro-organisms. I may say I do not intend to deal with those poisons.

Now, in order to prevent poisoning, there has been legislation, and I daresay legislation does prevent poisoning to a certain extent, and it is certain that now for some few years past the number of cases of criminal poisoning which have been the subjects of judicial investigation have been singularly small. In order to prevent poisoning, the law tries to make it difficult to get

a poison without having the fact registered. The sale of poisons is regulated by an Act of Parliament, and this sale of poisons applies to people who keep an open surgery, and of course it applies to druggists. By this law, articles which are to be deemed poisons are divided into two classes. Part 1 comprises arsenic and its preparations, prussic acid, cyanides of potassium, and of metallic cyanides, strychnine, and all poisonous vegetable alkaloids and their salts, aconite and its preparations, emetic tartar, corrosive sublimate, cantharides, ergot, &c., these latter being put in to meet the crime of abortion. Part 2 comprises oxalic acid, chloroform, belladonna and its preparations, the essential oil of almonds (unless deprived of its prussic acid), opium, poppies, and their preparations. You will note that Part 1 contains more deadly poisons than Part 2. On the sale of any poisons contained in Part 1 or Part 2, the box, vessel, or cover in which it is contained must be labelled with the name of the article, the word "poison," and the name and address of the seller. No poison in Part 1 may be sold to any person unknown to the seller, unless introduced by a person known to the seller, and upon every such sale the seller must, before delivery, enter into a book, to be kept for the purpose, the date, name and address of the purchaser, the name and quantity of the poison, and the purpose for which it is required; and he shall also cause the purchaser to sign his name therein. Therefore, you cannot get a deadly poison without considerable difficulty. A penalty of £20 may be inflicted upon any one not conforming to the necessary regulations. Poisons must not be sold by people who have not passed a qualifying examination in pharmacy or medicine. A year or so ago a case happened which was of interest in this connection. A druggist went away for the afternoon on some private business, and he left a boy in the shop. While he was away somebody came into the shop, whom the boy knew, for a packet of vermin killer, 3d., containing strychnine. He sold it to him after going through all the necessary formalities, but the chemist was fined £5 because the boy was not qualified.

But the law may be evaded. One noted instance of this was Eliza Edmunds, of Brighton, who gave strychnine to a medical man in chocolate creams. Eliza Edmunds got her strychnine in the

following way:—She waylaid a little boy in the street, and said, "I wish you would go to Mr. So-and-so, the druggist, take that note, and bring back to me the answer." The letter pretended to be from a doctor in Brighton, and was to this effect: "Will you kindly let bearer have so much strychnine; I am suddenly quite out of it. Signed, ——" The strychnine was sent, and so Eliza Edmunds got the poison. I take it that if a person is determined to get a poison, in a case of deliberate murder, he or she will find out a way. But undoubtedly the Poisons Act is of very great value in giving an hot-tempered, impetuous sinner, a little time for reflection.

Toxicology is the science of poisoning, and I need hardly remind you that the word comes about in a roundabout way. It is derived from *toxon*, a bow, because poisons were first used upon arrows. Allusions are made to poisons of this kind as early as Homer, and throughout ancient history you find constant reference to poisoning. Poisons have been used a good deal for the execution of criminals, and a noted instance was that of Socrates, whose case I shall read to you when we come to it. He was poisoned by conium or hemlock, which has been largely used. At the present day we hear a good deal about euthanasia from a certain class of writers, who contend that it is proper and advisable, when people are hopelessly diseased, that some painless form of death should be provided for them. It is reported that the old men of Cos, when tired of life, used to assemble at a banquet and drink a happy dispatch to each other in draughts of hemlock. Poisoning, therefore, is not a new thing. Poisons were common in the ancient days, and people in eminent positions were liable to poisoning. It is said that Hannibal always carried poison with him, in order that he might dispatch himself if he got into trouble with his enemies. Mithridates, king of Pontus, earned great fame because he invented a universal antidote. If anything were wanted to show that poisoning was common, it would be the fact that there should be need for a universal antidote. Amongst early criminal poisonings we find it on record that Parysatis, the wife of Darius, killed Stalira, the wife of Artaxerxes, by carving a fowl with a knife smeared with poison on one side only. That is a fact which shows that the higher education of women is no new thing. As early

as B.C. 330 there is an account of a society of women who took to poisoning obnoxious senators of Rome. Nero retained the services of a lady named Locusta, whose duty it was to do his poisoning for him. Amongst the poisons aconite was frequently used, and there is a line in Juvenal in which Pontia, who killed her two children, is represented as saying, "Confiteor puerisque meis aconita paravi." With the advance of chemistry and the preparation of new bodies poisoning became more general. When white arsenic became tolerably common there was an increase in the amount of poisoning. It is said that some of the poisonings early in the seventeenth century in Italy were done by white arsenic; and some of the poisoners in Paris, such as the Marquise de Brinvilliers, who is commemorated in one of Dumas's novels, used white arsenic. You will find that with the advance of chemistry the poisoner has been quite ready to use the latest new drug. One cause of the decline of poisoning is that the analytical chemist has been able to cry "Check" to the poisoner. All the bodies are detected with greater ease now than formerly. When strychnine was discovered, there is no doubt it was used many times without being detected. The cases were regarded as tetanus, but at the trial of William Palmer the whole question of the detection of strychnine was thrashed out, and that marked the practical cessation of the use of strychnine as a poison. Lampson, another medical poisoner, used aconitine. It was thought that that could not be detected chemically. It is said that the chemical tests for aconitine are very doubtful. Quite true, but the physiological tests are of another kind, and Lampson's crime was brought home to him, and the analyst was able to again say "Check" to the criminal. Therefore the knowledge of the analyst has been of great value in arresting the growth of criminal poisoning.

An Agreeable Form of Administering Trional.—According to Dr. Habermann's observations ('Allgm. Med. Central-Zeitung,' No. 32, 1898), the exhibition of trional in a carbonated alkaline water facilitates its absorption, renders it more agreeable to take, and produces hypnotic effects in much smaller doses.—*Philadelphia Polyclinic.*

THE PATHOLOGY AND TREATMENT OF CHRONIC VARICOSE ULCERS OF THE LEG.

By ERNEST LAPLACE, M.D.

CHRONIC varicose ulcers of the leg form a class of cases which not only haunt the hospital wards, but are a burden and lasting reproach to the surgeon. Only those who have dealt with a great number of these cases can realise the amount of careful treatment they demand in order to reach any degree of improvement, and only these can realise the disappointment and discouragement that follow a return of the trouble soon after the improvement. In order, therefore, to apply the rational treatment to these rebellious sores, we must first consider their pathology.

The varicose ulcer is appropriately named such because it depends for its existence upon a serious disturbance of the superficial circulation of the leg. This disturbance results from the dilated and weakened condition of the veins, producing an amount of stasis, distending the skin, causing an œdema, which requires a very little traumatism to produce an excoriation, which is the starting-point of the ulcer. If we inquire as to the initial cause of varicose veins we are confronted with a very obscure problem in pathology, and can only say that the walls of these veins are weakened by a certain process of mal-nutrition, resulting from an impaired condition of the whole system. The veins are weakened most probably in the muscular coat; the column of blood which they are intended to support excites a stronger pressure within their calibre than they are able to withstand, and as a result dilatation ensues. This dilatation renders the valves of the various veins almost useless in supporting the column of blood, and the veins acquire at times an enormous distension. An erect posture, and especially such vocations as require constant standing, tend to aggravate the pathological conditions just described, and as a result, from the time a slight traumatism or excoriation has removed the epithelium from the passively congested limb, an ulcer exists whose tendency is, by the impairment of the circulation, to grow constantly larger, and to be accompanied by venous hæmorrhages.

The effort at repair on the part of nature immediately results in the formation of granulations

and cicatrisation. On the other hand, infection sets in, and suppuration complicates the case. In the acute stage these ulcers are soft and tender, and rather hæmorrhagic; but after having existed for months or years the chronic irritation of the edges of the ulcer and the neighbouring parts results in the formation of fibres within the surrounding cellular tissue, whereby a hard callous condition ensues. This hardening further impairs the circulation, so that in time the varicose ulcers possess a dull pink, glazed appearance, showing a great lack of blood about the parts. This scarcity of blood is due to the compression of the neighbouring parts by the chronic inflammation which accompanies the ulcer.

For years, in hospital and private practice, I have given special attention to the treatment of these ulcers. The chief dependency has been the application of the modern principles of asepsis, combined with rest. Having treated the patient so as to improve his general condition, I proceeded under ether to remove freely the edges of the ulcer and scrape its surface, transforming the ulcer into a healthy wound. The patient was then kept at rest in a recumbent position so long as was required to entirely heal the ulcer. The wound was dressed at proper intervals so as to maintain it constantly in an aseptic condition. In a majority of instances, by dint of great efforts, I succeeded in healing the ulcer. The patient would be advised to wear an elastic stocking, and to keep off his feet as much as possible. It was impossible in many cases to follow this advice, inasmuch as most of the patients belonged to the labouring class. As a result, from use of the limb, I often found that the place where the ulcer was soon became congested, blue, and swollen, reproducing the same old ulceration. It is in these cases that both patient and surgeon feel so discouraged.

Analysing these unfortunate cases, I find that the treatment of the ulcer was based on the proper principles. I removed the cause of the ulcer by placing the patient in bed, eliminating the passive congestion which accompanies the varicose vein. The ulcer was healed by removing the infectious element and transforming it into a healthy aseptic surface, whereon nature was enabled to build fibrous tissue and cover it up with epithelial cells. So far everything was correct, and the indications of treatment were properly fulfilled. But from the

time that the patient was allowed to walk again, and the same original cause was again at work, it was natural that the effect should also be the same, and show itself in the return of the ulcer. The weak point in the treatment, therefore, was the fact that the varicose veins and their effects were not permanently eliminated. I believe that the removal of the cause—that is, the cure of the varicose veins—should be the initial step for the successful treatment of these ulcers, and therefore advise that all the veins of the leg be ligated and obliterated as the first step in the treatment. The treatment of varicose ulcers of the leg, acute or chronic, to-day should resolve itself into a radical cure of varicose veins of the leg, and as a result the ulcer will immediately take on a tendency to heal, and will require only ordinary attention to help the healing process. Furthermore, the cause being removed in the complete obliteration of all the superficial veins, the ulcers once healed remain permanently so, allowing the patients to attend to their work with impunity.

For the obliteration of the veins I have performed one of two different operations, according to the nature of the varicose veins. In some instances the superficial veins of the leg are persistently dilated, but the long and short saphenous veins are not distended. In this case a catgut ligature is applied to these veins as they enter respectively the saphenous and popliteal veins. This method was advised by myself in the year 1892, and has met with success in this class of cases. Inasmuch as these two veins drain the whole of the venous circulation, their ligature results in a simultaneous stasis and coagulation of blood within all these superficial veins. Their obliteration soon follows.

On the other hand, when other great masses of enlarged veins form on the limb, and do not affect the saphenous vein above the knee, I practise Schede's method, which consists in making a circular cut about two inches below the knee, and directly through the skin, separating all the veins as they present themselves, clamping them, and subsequently ligating them with catgut. The circular cut is then carefully sutured. This is the most effective method of stopping the superficial venous circulation, and obliterating the veins which hampered the process of healing.

These wounds, after the obliteration of the veins,

heal most kindly when thoroughly aseptic, and from the first the operation exerts a beneficial effect upon the ulcers. They immediately acquire a more healthy appearance, and granulations set in which effectually build tissue, while the surrounding epithelium grows and covers the ulcer, completing the healing process.

The combination of an operation for the obliteration of the venous circulation and the proper local treatment of the ulcer according to modern surgical methods fulfils all the indications in this rebellious class of cases. Since I have adopted this method I have obtained very flattering results in the most obstinate, old, and callous ulcers at the Philadelphia, Medico-Chirurgical, and St. Agnes's hospitals during the last three years. A series of eighteen ulcers of the worst varieties healed and remained so. A detailed description of the cases would be rather tedious, since the principles involved in each one are precisely the same, and only require such modification as the peculiar location and character of the sore may demand.

I believe that syphilis and tuberculosis may complicate at times these varicose ulcers, hence we should never lose sight of the diathesis under which the patient may suffer, so as to eliminate it by the proper treatment before the surgical aspect of the case is undertaken. In syphilitic ulcers accompanied by varicose veins we have given a general mixed specific treatment, in addition to a local application of a salve consisting of ammoniated chloride of mercury five grains to one ounce of vaselin.

When the sore had taken on a healthy appearance, and the tissue seemed comparatively free from specific poison, the operation upon the varicose veins was undertaken—not before. In tuberculous ulcers complicated by varicose veins a general tonic treatment was given, consisting of the syrup of hypophosphites, and a local application of iodoform powder. I always take care, however, to first remove all diseased tissue by the curette, and paring the edges of the sore. When the healing process, after the wound had become healthy in character, seemed slow, threatening to take a long time before the epithelium would spread over the edges and cover the surface, skin-grafting was practised, according to Thiersch's method. The granulating surface was thereby covered thoroughly with epithelium, and in the

course of two or three weeks would be completely healed.

This process of skin-grafting consists in removing large strips of epithelium from the thigh in the following way:—Having rendered the strip aseptic it is made tense by being supported between the thumb and the middle finger of the left hand; using an ordinary razor or sharp knife, the instrument put at an angle of 20° to the thigh, and practising a see-saw motion, the epithelium is, if possible, removed without penetrating below the papillary layer of the skin. These strips of epithelium may measure one fourth to one half inch in width, and three, four, or five inches in length, according to the necessities of the case. Aseptic gauze is applied to the spot whence the epithelium was removed, and secured by a bandage; this will suffice as treatment for this exco-riated surface. The spots of epithelium are made to cover the ulcer completely, and are secured in position by several thicknesses of sterilised gauze smoothly laid over the parts.

Should suppuration, however, set in, and the life of the grafts be in danger, this danger is overcome by the various layers of the gauze having been made detachable, so that when the dressing is removed all the layers of gauze can be taken away except the lowermost layer. In this way the growths are not disturbed. The secretion and pus can be removed effectually through this lowermost layer of gauze.

Peroxide of hydrogen will clean the wound thoroughly through this one layer of gauze, and the application of any antiseptic solution is made possible without disturbing this last layer. In this case it becomes necessary to repeat our antiseptic washing at least every day. At the end of a week or ten days it will be found that all of the grafts have adhered, whereas, should all the dressings have been removed when there was evidence of suppuration, usually on the third or fourth day, these grafts would have been sacrificed.

I believe this to be a very practical point, essential to success in a great number of cases. From experience I have found that it is not always possible to devote our best endeavours to maintaining an aseptic condition of the ulcers, especially if they have been grafted. A light bandage applied to the limb for a short while before the patient is allowed to get up suffices to complete

the treatment of what has for a long time been a very distressing condition, both for the patient and for the surgeon.

Therapeutic Gazette, September, 1898.

"IMMUNES" AND YELLOW FEVER.

From the *Charlotte Medical Journal*.

In a leading article on the United States Marine Hospital Service and Yellow Fever in our issue for August 20th we stated that we were somewhat sceptical as to one attack of yellow fever always insuring immunity against a further attack of the disease ('N. Y. Medical Journal,' Sept. 3rd, 1898). We quoted, moreover, two cases—one of a British army medical officer who contracted the disease a second time on returning from a visit to Europe, the other of an American who was attacked by the disease three times. We further prophesied that light would be shed upon the reputed value of this immunity by the regiment of "immunes." The issue of the 'Medical News' for the same date says that the fact that a number of men, who had survived an attack of yellow fever, enlisted and went to the front with the full assurance that they could face the dread disease with impunity, have been attacked with yellow fever, and some of them even died from it, has put a new phase upon the significance of the word immune. This journal further states that it is quite possible that life in a northern climate so renovates the system after an attack of yellow fever as to interfere with the proper balance between the toxins and anti-toxines, and so destroys the security against a second attack. This was mentioned by us as being the case with the British officers referred to. We have no doubt that relative immunity in some degree is conferred by one attack of yellow fever; but we assert once more that it is only relative, and we cannot but emphasise our exception to the universality of surgeon Murray's assertion, in the Government monograph on 'Yellow Fever,' that "one attack always insures the afflicted one that he is hereafter immune."

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SOME CONSIDERATIONS PRELIMINARY TO THE STUDY OF DYSPEPSIA.*

BY

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GENTLEMEN,—I take it that I shall meet with but little opposition if I assert that there is no department of practical medicine or group of clinical phenomena which are investigated, described, and considered with a greater vagueness and looseness of thought and method than the subject of indigestion. It is not that there is in the diseases of the circulatory apparatus, of the respiratory organs, or even of the nervous system less of which we are ignorant, but in connection with these and other functions what we do know is tolerably clearly defined from what we do not know, our ignorance no less than our knowledge is formulated, and, so to say, we know where we are. Next to positive knowledge, a clear recognition of what is not known is the most valuable aid to the progress of any branch of inquiry, and particularly so in one of so practical and complex a character as clinical medicine. In respect, however, to the maladies dependent upon errors in digestion, a singular lack of precision in thought and language appears to be all but universally prevalent. Nor does this seem to be quite justifiable, since on the one hand our knowledge of the normal physiology of digestion is as well advanced as is that of the other functions,—more so, indeed, than of some,—and on the other hand the frequency with which digestive disturbances occur offers the fullest opportunity for study if pursued on lines which have proved so fruitful in other directions of pathology.

I would at once desire to disclaim all pretension to an accuracy that is not possessed by others, but

* A Paper read before the Medical Society of London, October 10th, 1898.

I am merely stating as a fact what I believe would be at once admitted; and for example of my contention I would refer to our text-books and systems of medicine and ask any one to compare the mode of handling the sections devoted to diseases of the digestive organs with those of other functions. In the latter we have for the most part an attempt at logical treatment of the subject. The causes, symptoms, morbid anatomy, and other aspects of the several diseases are treated on a more or less uniform plan; facts are stated, provisional hypotheses are recorded, and a coherent and sequential view of each disease or group of diseases is presented, from which the inquirer may learn what is known and perceive the directions in which further information is required; in other words, there is an orderliness in the treatment of the subject which has been the outcome of systematic observation and scientific uniformity of plan. But if we turn to the chapters which deal with disorders of digestion or the diseases of the organs of digestion, there is at once apparent a departure from the method which obtains elsewhere, and confusion takes the place of order; the causal relation of symptoms seems to be imperfectly grasped, the limitations of the digestive process as distinct from absorption and other factors of nutrition are disregarded, and the manifestations of structural disease in the organs concerned are mistaken for the evidences of errors in the chemical changes involved.

Again let me say that my presumption does not extend to asserting that I am prepared to set the whole question straight; but if I can first of all gain your agreement that I have fairly stated the case, it will be a step towards the rectification of the position and dispose you more favorably to consider this my contribution thereto.

It is a poor pursuit, I am fully aware, finding fault with the language of any branch of natural science. It is inevitable that as the science progresses, the denotation of the terms and significance of its nomenclature must vary, until it is only by arbitrary consent they come to possess an agreed-upon meaning. And this difficulty doubtless is met with in no small degree in respect to digestion disorders. But there would seem to be a reluctance to employ many words in their legitimate sense, and still more to use them with any consistency. I shall make my position clearer and

render your criticism of my remarks the easier if I set forth the sense in which I employ some of the words I use, and this will be no peculiar or special use of them, merely such as they legitimately carry.

And first let me say that to my mind the very word digestion, and still more its correlative indigestion or dyspepsia—for they are synonymous—are apt to be employed with much uncertainty or at all events variability in their range of meaning. That digestion is a function of the body concerned with the food, and upon the maintenance of which the bodily health depends, is generally accepted; but of the scope of the function most different opinions appear to be entertained by medical men, with corresponding confusion as to the real nature of its disturbances. Surely it is advisable in this as in other functions to follow the physiologists in regarding digestion as a term denoting the entire series of changes which the ingesta undergo during their passage along the alimentary canal from their entrance in the mouth to the expulsion of the undigested, and unabsorbed residue. If this be the proper meaning of digestion, it is obviously improper to restrict the idea of it to those changes which occur in the stomach only, and if it may be said "Surely no one ever takes such a view," I can only say that in the practical application of our physiological knowledge to disease, the word indigestion or dyspepsia is constantly so limited. "Dyspepsia is a general name which is generally used as synonymous with indigestion" says a distinguished member of our profession in a standard work recently published. "It properly means discomfort and pain during digestion in the stomach, but it is frequently used to signify discomfort or pain during the process of digestion whether in the stomach or intestines. The vagueness of its significance has both advantages and disadvantages. The disadvantages of the name are that it signifies so many different conditions, that it is little or no guide to the exact functional or organic lesions of the several organs concerned in the digestive process; and without a more or less accurate knowledge of the nature of a disease we cannot hope for success in treatment. Its advantages, on the other hand, are that it includes a group of symptoms which cannot always be referred to an exact physiological and anatomical basis. In this

respect it corresponds to albuminuria or jaundice, affections which, although they can often be assigned correctly to their causes, yet sometimes defy exact diagnosis."* To my mind a more unscientific justification for the use of a word in a wrong sense is scarcely to be imagined. Its use is advocated on the ground of ignorance, which I would contend this very use helps to increase. But the quotation illustrates the current view that indigestion is essentially a stomach affection, and imperfections in the entire range of processes in the mouth and in the intestines are relegated to a wholly subordinate position, scarcely considered in fact. Now of the four successive stages of digestion, the oral or salivary, the gastric or peptic, the pancreatico-biliary and the intestinal, following one after another as they do, it is probable that the gastric is far from being the most important, its deficiency being made up for at other stages.

The simple object of the process of digestion, and so far as we know all that is attained by it, is the conversion of certain ingredients of the food into a fluid and diffusible condition whereby they may be absorbed into the blood or lymph currents, they of themselves as taken being wholly or almost wholly unfit to be so absorbed without undergoing digestive changes. I say "almost wholly," for there is some reason to believe that a very slight degree of absorption can take place even of those substances which ordinarily undergo considerable digestive change, as, for example, the beneficial results following nutritive enemata. It is the proteid, fatty, and carbohydrate elements of the food which specially require digestion, and of these the first-named are chiefly acted on in the stomach, the salivary digestion of the starches and the splitting up of the fats proceeding only to a very partial extent in that organ. But although the stomach is thus mainly concerned with proteid digestion, it is to be observed that the pancreatic secretion is equally if not more capable of effecting proteid conversion into peptones, and what is of equal importance to the present questions very little if any absorption of the digested proteids takes place from the stomach. Without going so far as to say that under ordinary circumstances the stomach is superfluous, physiological considerations would prepare us for the statement that its func-

tions may in great measure be compensated for elsewhere in the canal. Experiments on animals have gone far to show that this is so, and the recent remarkable case of complete extirpation of the stomach recorded by Dr. Carl Schlatter* has conclusively proved that the stomach is not an essential organ, inasmuch as the patient within a few weeks of the operation was taking without discomfort an ordinary dietary consisting of meat, bread, eggs, milk, &c., and a careful investigation of the metabolic products of the patient as compared with the ingesta, conducted by Hoffmann in Eichhart's clinic,† demonstrated a state of complete nitrogenous equilibrium, the patient gaining weight. The particular value of the stomach as a receptacle for receiving the food and passing it on into the intestine, portion by portion, and the use with which the hydrochloric acid of the gastric juice is credited as a guardian from bacterial infection and a controller of putrefactive fermentation in the intestine, are alike discounted by the history of this extraordinary case. Surely, then, in view of this quite subsidiary action on the part of the stomach, it is quite indefensible to regard the gastric digestion as the most important stage in the whole process, and that dyspepsia of other than gastric origin is practically non-existent. I feel that the preliminary step to a more rational understanding of indigestion is so to enlarge our views of the comprehension of this term as to make it coterminous with digestion proper, and recognise the occurrence of real duodenal and intestinal dyspepsia, more frequently than we are in the habit of doing.

Again, it is most desirable that the conception of "digestion" should be strictly limited to the process as above defined, and that it should not be taken to include the function of absorption. This is wholly different in its nature from the complex chemical changes which constitute the former; and whilst it is the second stage in the progress of the ingested food-stuffs towards the nutrition of the tissues, there for the first time are the digesta brought within the range of living protoplasmic activity as they traverse the columnar epithelial cells of the intestinal mucosa. The two functions have little or nothing in common in their dealing with the nutrient material, and as the

* Allbutt's 'System of Medicine,' vol. iii, art. "Dyspepsia," by Dr. Lauder Brunton, F.R.S., p. 385.

* 'Lancet,' January 15th, 1898.

† 'Munch. med. Woch.,' May 3rd, 1898.

physiologists consider each separately yet successively, so should the physician refrain from confusing the two, and endeavour so far as may be to ascribe to each the various symptoms which his dyspeptic patient may present. I am fully aware that defects in the due absorption of the digested material may become a determining cause of indigestion itself, just as an imperfect digestion of ingesta may interfere with their proper absorption; but this interrelation does not justify the processes being confused, or their limits disregarded.

There are other points also connected with the exact scope which the term digestion implies, which, to judge from the general character of writings on the subject, are often forgotten, especially when it is a perversion of the function which is under consideration; and one of these is the relation which the process bears to the digestive organs. This is peculiar, and has no counterpart in the animal economy. The function of digestion is a combined motor-secretory process, and the organs by which it is performed are muscular and glandular, the effective agents of each being specialised protoplasmic cells. The food-stuffs are propelled along a lengthy tube, and mixed with the secretions by muscular contractility, and the secretions successively perform their physico-chemical effects on them in virtue of the enzymes and acid or alkaline substances which they contain. But the point is that these changes take place outside the tissues, although in the body. We see nothing like this in the other functions whereby the bodily nutrition is maintained—absorption, circulation, respiration, and excretion,—and still less in the activities of the nervous system. As productive of disease there are, therefore, two very different conditions to be observed: on the one hand, disorders of the secreto-motor apparatus—structural, nervous, or vascular—by which the functional capacity of the apparatus may be interfered with; on the other, errors in the physico-chemical change taking place in the digesting material. It is true that these necessarily react upon one another, that imperfections in the digestive process, due, may be, primarily to errors in diet, may determine structural disease of the digestive organs, just as this latter will lead to mal-digestion. But it is very desirable to recognise their essentially distinct character, and the fact that each may exist to a certain degree independently

of the other in the production of what are known as dyspeptic symptoms.

One advantage of having this aspect of the subject in mind is that it furnishes a rational basis for classifying the causes of indigestion.* This subject I have elsewhere treated of, and it is sufficient for my present purpose to mention that the immediate causes of dyspepsia are—

1. *Errors in diet*, whether of quantity, quality (including irritant and toxic substances), or in the form in which the food is presented.

2. *Diseases of the digestive organs*, whereby their motor or secretory functions or both are impaired. Such diseases are:

- i. Structural—inflammation, degeneration, new growths.
- ii. Impaired nervous control, whether of central or reflex origin.
- iii. Abnormal blood and lymph vascular supply.

3. *Improper bacterial action* in the gastrointestinal contents.

4. *Defective absorption* of the digesta.

5. *Abnormal intestinal excretory processes*.

I am not concerned now to discuss these causal conditions, upon each of which much might be said, even of the last-named, which I think promises a not unfruitful direction of inquiry.

But I would point out that whilst all these abnormal states produce in a greater or less degree disturbances in the physico-chemical processes which take place in the gastro-intestinal contents, and as such are the cause of dyspeptic symptoms, the second of these, viz. the diseases of the organs, may give rise in addition to symptoms and signs expressive of these diseases—for instance, malignant disease of the stomach, besides the dyspepsia it may determine by its interference with the motility of the organ and the grave alteration it causes in the composition of the gastric juice, may lead to hæmatemesis, gastro-ectasis, &c., which are not evidences of indigestion, though due to diseases of the digestive organs. And further, inasmuch as the very process of indigestion may and does set up disease in the organs, certain of the complex symptoms which a patient presents are referable to this secondarily induced morbid state. Theoretically, therefore, these causal conditions give rise to disease manifestations in these different ways: first, dyspepsia proper by mal-performance of the

* "On Some Relationships of Indigestion." Hunterian Society's Lecture. 'Lancet,' October, 1897.

digestive process ; and secondly, the symptoms of diseases of the digestive organs, which may be primary in origin or secondary to the existing dyspepsia.

I pass now to say a few words in general upon the symptoms of dyspepsia, conditioning my remarks upon the nature of indigestion as I have set it forth, and the two-fold origin I have just described. It is customary—and indeed I think no self-respecting writer on the subject would feel at liberty to refrain—to describe the symptoms of indigestion as “protean.” I am not aware that the term tends to elucidate the problems at issue, but having conformed to established ways I proceed to express the opinion that the actual clinical manifestations of dyspepsia are not so numerous as is often supposed ; that many are rather evidences of the condition to which the dyspepsia is due ; that others are such remote results as to be practically removed from the exciting cause ; and the remainder, which are directly to be associated with flaws in the digestive process, and therefore properly dyspeptic, are capable of being grouped under very few heads, which much facilitates their comprehension.

Although not strictly definable, it is none the less convenient to speak of them as local and remote, the first being obviously and directly connected with the digestive organs. They may be tabulated thus :

A.—LOCAL.

I. *Subjective sensations.*

Discomfort. Weight and oppression. Sinking.
Pain.
Nasty taste in mouth.
Disordered appetite.
Acidity. Heartburn.
Nausea.

II. *Objective phenomena.*

Eruptions.
Vomiting.
Flatulence.
Diarrhoea. Constipation. Abnormal stools.

B.—REMOTE.

Nervous.—Headache. Vertigo, giddiness.
Subjective affections of sight and hearing.
Drowsiness, heavy sleep, torpor, wakefulness.
Mental irritability or depression, hypochondriasis, melancholia.
Referred pains. Cramps.

Circulatory.—Palpitation. Cardiac irregularity. Intermittence. Syncope.

Cardiac pain. Angina.

Flushing of face. Coldness of extremities.

Respiratory.—Cough. Dyspnoea. Sighing.

Cutaneous.—Various urticarial and erythematous rashes.

Urinary.—Deposits of urates, phosphates, oxalates, uric acid.

Albuminuria. Glycosuria.

Presence of aromatic sulphates.

Such a list—and I do not pretend that it is absolutely complete—is, I admit, a long one, and as varied in manifestations as it is lengthy. But it is not this multiplicity of signs and symptoms collectively denominated dyspeptic which, to my mind, makes their investigation difficult, so far at least as their reference to cause, and hence diagnostic value, is concerned, but rather the fact that many totally different conditions give rise to similar symptoms. The aim and object which as clinical observers we set before ourselves when investigating a case or group of cases of disease is, first of all, by systematic procedure to ascertain as accurately as may be, with various instruments of precision and otherwise, the departures from the normal working of the body, and such alterations in gross structure as physical examination may disclose. Such evidence—the signs and symptoms of the disease—we proceed to consider in respect to their underlying cause, and from the evidence infer what is actually wrong—what, in fact, is the structural abnormality upon which the functional defects depend. In the course of making this inference or diagnosis we rely first and mainly upon our knowledge of the normal organism in its structure, gross and minute, and in its action ; secondly, upon the experience of what structural or organic changes have been discovered previously to be associated with certain signs and symptoms. This is the basis of procedure upon which we teach our students, and more or less consciously it is the plan we pursue in every patient we examine, however much we may jump to conclusions. It is in the application of this principle to the causal diagnosis of dyspepsia that the real difficulty in the problem lies, the fact that no single symptom is diagnostic—I will not say pathognomonic—of the cause, but that, as I have said, the same symptom may be expressive of various underlying states. Take, for instance, the question of pain ; how can that help us to ascer-

tain the nature of the cause in any given case of indigestion? and to do that is essential before any rational plan of treatment can be proposed, to avoid the unscientific routine of bismuth, acids, alkalies, bitters, pepsin, &c. Reference to the list of causes I have given will suffice to show that we must seek to differentiate between errors in diet and morbid states of the digestive organs; we may for the moment disregard the other and rarer causes of mal-digestion. This is the first and fundamental step in the procedure. A moment's reflection will serve to show that pain may follow something that has been taken into the alimentary canal, whether such an irritant as a metallic poison or some indigestible article of food. On the other hand, so distinct a structural lesion as an ulcer of the stomach or a growth in the intestine may be provocative of considerable pain quite apart from any special irritation that food may induce. Or, again, in conditions of the stomach where the actual disease is less obvious, the condition which is known as gastralgia, when the sensory nerves of the viscus are unduly susceptible to the reception of impressions of a painful character,—here pain is a marked, and indeed almost the only symptom. What, then, is the significance of pain as diagnostic of the existence of either of these morbid states? Nor can more reliance be placed upon its character, so inadequately described by the current adjectival terms, or the situation in which it is experienced, nor even always its relation to food, since in well-recorded cases of ulcer, relief has followed a meal—a circumstance that in the main does serve to distinguish a gastric ulcer from a gastric neuritis. Or again, nothing is more certain than that the digestive process may be most imperfectly performed without anything resembling pain being experienced; even a gastric ulcer has been found post mortem, the presence of which has not been suspected during life; whilst pain is commonly absent from many cases of defective motility of the gastro-intestinal musculature; and also where there is good reason to suspect secretory imperfections as a cause of mal-digestion; and serious dietetic errors may be long continued without more than the slightest sensory discomfort. It follows, then, that the presence or absence of pain cannot be taken as definite evidence of indigestion, far less as indicating with any approach to certainty the cause to which an indigestion is to be

attributed. And what is true of so pronounced a symptom as pain is also true of other dyspeptic manifestations; vomiting and constipation, for instance, are each referable to food, to obvious structural disease of stomach or bowel, or to nervous causes acting centrally or reflexly. And the same may be affirmed in respect to the remoter symptoms, which require first of all to be clearly recognised as dyspeptic in origin before the particular factor of the dyspepsia can be positively indicated,—such, for instance, as headache, anginous attacks, cough, deposits of urates in the urine, oxaluria, &c.

The main difficulty, I repeat, which attends the clinical diagnosis of dyspepsia is not the detection of the symptoms and signs, which for the most part are obvious and easily ascertainable, but it is the association of these symptoms when perceived with the morbid condition which determines them.

The difficulty also is further increased by the very small part that physical examination plays in the detection of disease of the alimentary viscera. Excluding the liver, an organ whose connection with digestion proper is far less important than formerly supposed, we really learn very little by the application of our senses of sight, touch, and hearing. Certain alterations in the size and situation of the stomach, abnormal distension of the colon, and a proportion of new growths connected with the organs in question may be recognised; but if we compare the range of application of these methods with what can be done in connection with the heart and lungs the disadvantage at which we are placed is at once obvious. To a very great extent we rely for our recognition of primary disease of the digestive organs upon the presence of symptoms of indigestion, symptoms which are equally indicative of condition in which the organs are little if at all affected.

And here it will be necessary for me to refer to the clinical varieties of dyspepsia as they are described in books and elsewhere.

So long as indigestion was considered to be a malady mainly, if not entirely, connected with the stomach, such forms as "irritative dyspepsia," "atonic dyspepsia," and the like, found a place if not a precise meaning; the phrases suggested a corresponding plan of treatment, by alkalies or acids, sedatives or tonics, by the success of which their use was justified. Clearly such names should be

discarded now, and as surely should those based upon the preponderance of one symptom, such as "flatulent indigestion." Whilst the continued use of such a term as "biliousness" or "bilious dyspepsia" can only tend to obscure the real state of the case, and may well be left to denote that condition of uncertain pathology which, as has been well said, is cured by blue pill. Far less open to objection is a classification based on the site of the digestive position, oral, gastric, duodenal, intestinal. Though the data for such a grouping are still very deficient it is certainly an end to be kept in mind in the consideration of the subject from a diagnostic point of view. From another and correlative aspect to this the forms of dyspepsia may be grouped on the particular element of the food which is ill digested, such as proteid indigestion, fat indigestion or carbohydrate indigestion. A complete knowledge of the subject, which we are far from possessing, would enable us to go far in this direction. Probably the most satisfactory plan on which to found a classification is that based on causation—the immediate causation, that is, such as I have described. This arrangement would give the following forms:

1. Dietetic.
2. Symptomatic, *i. e.* the manifestation of primary affections of the digestive organs, structural, nervous, and vascular.
3. Bacterial indigestion.
- 4 and 5. The rarer conditions of indigestion due to defects in the due absorption of the digesta, and to improper excretory processes taking place in the intestine, probably occur so infrequently—at least apart from other causes—as scarcely to require special naming, but the terms "absorptive" and "excretory" dyspepsia are consistently suitable.

The full application of such a scheme would involve the consideration of the locality in which the indigestion is taking place—mouth, stomach, or intestine; and would also of necessity take note of the kind of indigestion, whether proteid, fatty, or amylaceous. But whatever plan be adopted, the correlation of symptoms and cause will still remain a desideratum, and until that has been worked out any arrangement can only be provisional. I would, however, claim for the one proposed a nearer approach to scientific precision than any in current use.

Not the least among the advantages that would follow, as I conceive, the adoption of some such plan as this would be to furnish a clearer conception of the relation of the several diseases of the digestive organs to indigestion, and to doing away with such meaningless, and I would add mischievous terms as "functional disorders" of the stomach or bowels. All disorders of the organs concerned in the digestive process which interfere with the proper performance of that process are functional disorders as much as they are structural diseases. The existence of the one implies the other. The deficient motility and altered secretion which characterise an acute gastritis or a gastric cancer are as surely the causes of indigestion as the less obvious, but none the less existent structural change which determines the motor secretory imperfections in so-called "gastric irritation" and "gastric inadequacy," or even "nervous dyspepsia," which may be taken as examples of "functional disorders of the stomach." How far these conditions can exist without departures from the normal structure of the organs concerned, however slight and transient these departures may be, I confess I am at a loss to understand.

"Gastric irritation," says Dr. Sidney Martin,* "may be defined as a functional disorder of the stomach in which there is irritation of the organ, produced by the food itself in the form in which it is swallowed, or by the food in the process of digestion; it may be primary (*i. e.* occur in healthy individuals), or it may be associated with or predisposed to by certain diseases or general conditions of the body. Food bears to gastric irritation and to gastric catarrh the same relation that irritating particles and gases bear to bronchial irritation and bronchitis." I should myself have thought that bronchial irritation was as much a structural imperfection as bronchial catarrh or bronchitis, and, *mutatis mutandis*, gastric irritation as gastric catarrh, and that the altered function (disease) was as much an expression of altered structure in the one as in the other. I have on a former occasion† criticised the expression "functional disease," and need say no more, except that if there ever exists a condition

* 'Functional and Organic Diseases of the Stomach,' by Sidney Martin, M.D., F.R.S., 1895.

† 'Westminster Hospital Reports,' vol. ii, 1886, "Functional Diseases."

in which the digestive organs are quite healthy, and the secretions and motility of the canal normal, and yet, owing to the nature of the food, some mal-digestion of that food takes place without the organs or their functions being affected, then there would be a purely functional indigestion unaccompanied by structural defect; though how long such a condition could last, or whether it could be recognised, or whether, indeed, it actually could occur, I am not prepared to affirm; but that is an entirely different thing from a functional disorder of a viscus.

The bearing of the foregoing remarks, which appear to me to be among the necessarily preliminary considerations to a study of dyspepsia, would, perhaps, be better appreciated were I to take up somewhat in detail one of the conventional varieties of indigestion; and no one would serve my purpose so well as the so-called "nervous dyspepsia," a term that is more frequently on our lips than, I fancy, its real understanding warrants.

Most authors follow more or less closely the description of Leube, who nearly twenty years ago published a paper* on the subject, and in so doing gained the credit of being the first to differentiate it from other forms of chronic dyspepsia. The causal basis of his definition of the condition is the existence of such a general nervous state as hysteria or neurasthenia; and practically it comes to this, that dyspeptic symptoms occurring in such patients, with the singular proviso that the digestive act, so far at least as the stomach is concerned, is accomplished within the normal time as ascertained by a test meal, constitute nervous dyspepsia. This latter circumstance is an essential feature of nervous dyspepsia according to Leube, but it is very doubtful whether such a position can be maintained. Apart from that the reference of the condition to a fundamentally underlying nervous cause is satisfactory, though not, perhaps, wide enough as originally laid down. Dr. Sidney Martin, who from the point of view of stomach only, adopts in great measure Leube's view, also says: † "There are many cases of gastric irritation, and, indeed, of other disorders of the organ (gastric insufficiency), in which certain symptoms attain such prominence as to give a feature to the

particular case. Such symptoms are without exception referable to some change in the nervous system, and are by some included under the terms nervous dyspepsia and neurosis of the stomach." In this case the limitations of the malady are conditioned by the symptoms rather than by the cause. Or again, Dr. Goodhart tells us that "although there are all sorts of different dyspepsias elaborated out of the heads of individuals, if I were going to write a book on indigestion I should first devote myself to a volume on diseases of the nervous system. Surely," he continues, "I cannot be wrong when I make the statement that of the number of dyspeptics that fill the doctors' lists the great proportion by far are neurotics in one form or another. It is really no great exaggeration to say that there are only two forms of indigestion; that produced by over-eating and drinking, and that due to failure of nervous power. There are other conditions that are met with as rarities, but these who knows anything about? Then we talk about acid dyspepsia and atonic dyspepsia, as if any dyspepsia were other than acid, and from my point of view as if atonic dyspepsia were necessarily something different." ("Common Neuroses." The Harveian Lectures, 1891). With all submission there seems to me a good deal open to question in these statements, but as bearing upon my present purpose, viz. nervous dyspepsia, it is clear that the distinguished writer regards it as a very widely extended malady, far more so than the other authorities I have quoted. Possibly, as his theme was "common neuroses," he had been regarding disease at large through "nervous" spectacles. In marked contrast is the opinion of a later author,* who states that "in nervous dyspepsia there is an increased irritability of the stomach nerves, especially the sensory. Whether this increased irritability is really due to a mild inflammatory process in the mucous membrane is difficult to determine. In the neurasthenic a slight degree of inflammation not recognisable by our present methods could produce morbid manifestations in the form of a dyspepsia, which only an advanced gastritis would otherwise call forth." And he concludes that nervous dyspepsia is a disease by itself, and chiefly a sensory neurosis, though the motor and secretory functions may also be impaired in it; that it is not so frequent

* "Ueber nervose Dyspepsie," *Deutsch Archiv f. klin. Med.*, 1879, vol. xxiii.

† Loc. cit., p. 208.

* Rosenheim, *Berl. klin. Woch.*, Nov. 1, 1897.

a disease as has been assumed; and that though it usually exists along with other nervous manifestations it is wrong to assume it as a symptom of neurasthenia. My object in submitting these quotations—and they are but a sample of many—is to show the extreme diversity of views that is held upon the nature and scope of a malady that we are constantly in practice speaking of,—a diversity, I would maintain, that is due to the generally vague and indefinite notions that prevail upon the real meaning of indigestion. Throughout these opinions there runs, more or less expressed, the idea that indigestion is a gastric perversion only, and in some cases symptoms, and in others underlying nervous states, are made the basis for the separation of the particular variety of dyspepsia. I much doubt whether any not infrequent disease of any other system would be so vaguely dealt with, or that an inquirer would gain such little real information to help him understand the nature of the malady were he to seek for it from any of these authorities I have quoted, whilst the divergences amongst them would only mystify him.

By the adoption of some such arrangement as I have indicated the place and scope of nervous dyspepsia would be at once laid down, and, what is of equal importance, its relationship to other modes of digestive disturbance. It would be defined as that form of disturbed digestion due to impaired innervation of the digestive organs, whether mouth, stomach, intestines, or associated glands, and leading to imperfect changes in any or all of the different food constituents—proteid, fat, or starchy, according to the locality of the affection. The disordered nerve control would be due to, or, in other words, the causes of nervous dyspepsia would be—

a.—Disturbances of central origin.

Emotional states; anxiety; overwork.

Obscure conditions of the nervous system, such as hysteria, neurasthenia.

Gross structural lesions, such as cerebral tumour, meningitis, locomotor ataxy.

Toxic bodies.

b.—Disturbances of reflex origin, as from kidneys, ovaries, &c.

c.—Peripheral irritation of nerves of alimentary canal.

The term nervous dyspepsia, therefore, would
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not denote a group of symptoms, but an indigestion primarily of nervous origin.

The function of digestion consists, as I have reminded you, of a series of physico-chemical processes affected by various secretions, and aided by movements which mix and propel the gastro-intestinal contents; and indigestion is due to some flaw in one or more of these different acts, which causes what we speak of as dyspeptic symptoms. Now both the secreting and the motor factors of digestion are controlled by the nervous system; directly by influences on the muscle and gland cells, and indirectly by regulating the blood and lymph supply to these structures. It would be expected, therefore, that the symptoms of a nervous dyspepsia would be such as are directly traceable to a disturbed motility or secretion, or both, and such is actually the case; for example, the retching and vomiting that fright or anxiety may produce; the obstinate eructations or vomiting, perhaps after every meal, of the hysteric; the characteristic vomiting of cerebral tumour, or of the gastric crises of locomotor ataxy; or the circulation in the blood and nerve centres of certain poisons. Note also the arrested movements of the stomach which may attend emotional states, as may be seen by the undigested meal vomited long after it should have passed through the pylorus; or the increased intestinal peristalsis, leading to diarrhoea, from fright or other psychical states. These illustrate the perverted movements determined by impaired nerve control. Our knowledge of the innervation of the digestive glands is tolerably precise as regards the salivary and rather less so in respect to the pancreas, whilst little is known with any accuracy about the nerve governance of the gastric and intestinal tubules, to the activities of which the peristaltic movements of the gastro-intestinal musculature appear to be an important stimulus. We have, however, very distinct evidence that the gastric juice is liable to considerable alterations in quantity and quality, a marked increase or diminution of the hydrochloric acid, and perhaps also, though less certainly, of the pepsin. Such modifications are among the most marked phenomena of nervous dyspepsia, to which many of the more obvious symptoms of that affection are due. The local sensory manifestations are attributable to the disturbed motor secretory functions. It is seldom that severe gastric pain is

complained of, and such a symptom, when present, I regard as gastralgia due to a hypersensitive condition of the nerve terminals—a hyperasthenia comparable to that of certain areas of the skin noticeable in gastric ulcer,—and though not infrequently accompanying indigestion, is not itself any evidence of mal-digestion proper. Alterations of appetite are among the best known results of many nervous states, and all degrees of absence, excess, or capriciousness are frequent. The marked degree of perversion associated with extreme emaciation, known as *anorexia nervosa*, is due to a more profound disturbance than that of digestion merely; more probably the absorption of the digesta is defective, or the entire metabolism of nutrition at fault, since it is a wholly different condition from that following simple deficiency or even deprivation of food inflicted on a previously healthy person.

Flatulence, which in my experience is not a marked symptom of this variety of dyspepsia, is rather to be regarded as a secondary result of the improper digestion of amylaceous constituents, except in such cases as it is due to sudden and extensive escape from the blood-vessels into the intestine. Passing to the more remote symptoms—headache, dizziness, drowsiness or insomnia, mental irritability, hypochondriasis, irregular pains, cramps, cardiac palpitation and irregularity, coldness of extremities, &c.—it would be exceedingly difficult to say how far such manifestations are not due to the primary nervous disturbance which has been the cause of the indigestion. These and other symptoms of the same character, when they are clearly to be attributed to mal-digestion, are probably caused by the formation of toxic substances in the course of the digestive changes which are absorbed and circulated, or to variations in blood-pressure.

The last point to which I would refer is that no one of these symptoms is in any degree diagnostic; each one of them may occur in other forms of dyspepsia in which the nervous influence is not primarily perverted, if, indeed, it is at all. Vomiting, diarrhoea, and other motor disturbances are among the commonest evidences of an indigestion of dietetic causation or of gastritis, as likewise flatulence and the whole series of remote phenomena. Hyperacidity is met with under similar conditions as in gastric ulcer, whilst a deficient secretion of gastric acid commonly occurs in gastric cancer. As I said earlier in my

paper, it is that these various symptoms are none of them specially connected with any one cause, but are more or less common to all causes, that makes the ascertainment of the cause by study of the symptoms so difficult—the difficulty, indeed, which characterises the whole subject. But I do not regard such a difficulty as insuperable, though I am not prepared now with the solution; but I feel convinced that before any rational treatment can be propounded with reasonable hope of success, it is this problem which has to be overcome; and as preparing the ground for so doing by an orderly and scientific method on a physiological basis these observations are offered.

Gasoline Intoxication.—The following is the report of an accident by S. H. Starbuck, M.D.:

I was called this afternoon hurriedly to the house of a near neighbour to do what I could for four children thoroughly anæsthetised from inhaling gasoline. The mother had been using gasoline to cleanse some articles of household furniture. The children were in the room, with all the windows open, but they were playing on the floor near where it had been used. The mother stepped from the room for a very short time, and upon returning found all the children asleep upon the floor. The oldest one of the children is eight years, the youngest sixteen months. Breathing in all the cases was very natural. The pupils were contracted, skin pale and cool to the touch, the conjunctivæ insensible. The anæsthesia seemed to be complete, and lasted in the oldest child about five minutes after being removed, and with the other three for twenty to thirty minutes. They all awoke vomiting, and this evening are apparently all right.

I know of one case before these that happened a few years ago. A child of five years of age would upon every opportunity go to the gasoline stove, turn on the gasoline, and inhale it until he would become intoxicated.

Cincinnati Lancet Clinic, Sept. 17th, 1898.

CONSTIPATION of muscular origin in women, resulting from pregnancy and implicating the abdominal and perineal muscles, should not be treated by purgative. Symmetrical massage and gymnastic exercises are called for. The diagnosis is made by observing the feeble abdominal constriction.—*American Practitioner and News*.

THE REAPPEARANCE OF HERNIA AFTER THE OPERATION FOR THE RADICAL CURE.

A Clinical Lecture delivered at St. George's Hospital,

BY

WILLIAM H. BENNETT, F.R.C.S., &c.,

Surgeon to the Hospital, &c.

At a time when the treatment of hernia by the radical cure (so-called) is so commonly adopted as it now is, the question of the liability to recurrence or reappearance of hernia after the operation must necessarily be of some interest. That reappearance of hernia after operation does take place, not very infrequently, must, I think, be admitted, although the confidence with which some operators appear to regard the treatment would at first sight almost lead to the conclusion that recurrence was unknown. It must, however, be borne in mind that it is extremely difficult to follow up for long periods hospital cases, in which recurrence is much more prone to happen than in patients in the better classes of life, for reasons to which I shall presently refer. I could myself produce a considerable list of operations performed in private practice, some many years ago, in none of which has recurrence happened up to the present time, but an analysis of operations performed in hospital practice during the same period would certainly not be so satisfactory. The reasons for the different result in the two classes of cases are not obscure, as I hope to be able to show in the present lecture. As a basis for my remarks I must call your attention to two interesting cases of reappearance of hernia after the radical treatment which have recently been under my care in the hospital. One of these is especially worthy of study, as it demonstrates very clearly certain weak points in the operation for the radical cure, no matter how thoroughly it may have been carried out.

The first case is that of a patient aged 45, a big man, very stout and heavy, a signalman, an occupation entailing hard manual work. He originally came into the hospital about eight months ago, and I operated upon him for a very large inguinal hernia on the left side. The operation was diffi-

cult; the hernia was very large, composed of a small amount of gut, and an enormous amount of omentum, which was removed; the ring was so large that I could nearly pass my hand through it. The muscular structures were strong, and excepting the large size of the ring the case was by no means unfavorable for the application of the radical cure. I exposed the parts, freely laying open the external oblique to ensure the structures being thoroughly dealt with. The conjoined tendon, internal oblique, and Poupart's ligament were tightly brought together with stout silk, so that these structures were in close contact, the large ring being entirely obliterated with the exception of a small space through which the cord had to pass. I did not displace the cord upwards. The sac was previously isolated, ligatured high up, and removed.

The case did admirably, and the patient left the hospital at the end of the usual time, so far as we could tell perfectly cured. As he was anxious not to wear a truss I thought it a good opportunity for testing the radical cure, so we let him go home without a support. He continued at his work for some time, but about three weeks ago he noticed the hernia coming again. This is a good example of reappearance of a hernia after a thoroughly performed operation for the radical cure, done under good circumstances, in a subject more suitable for the operation, in spite of the large size of the ring, than many of those with whom we have to deal.

The man was so well satisfied with the first operation that the rather rapid return of the hernia did not prevent his coming back in order that the operation should be repeated. I therefore operated again a few days ago, and a most interesting condition was found. All that had been done in the first operation remained perfect; the conjoined tendon and Poupart's ligament were still held together by the sutures, and the cord passed through an opening of the size which would accommodate the cord under natural circumstances, so that in the true sense of the word there has been no recurrence of the hernia. The tension, however, which was necessary to bring together the conjoined tendon and Poupart's ligament, or the strain to which the parts were afterwards subjected, must have led to some splitting of the conjoined tendon, and a new hernia had made its way through the split in the tendon. There was, in fact, in consequence of the

operation, a hernia through the conjoined tendon—the variety described in the text-books as a direct inguinal hernia. The sac of this hernia had nothing to do with the old sac, the stump of which we found as we left it at the first operation, thoroughly obliterated and in good condition.

The special interest of this case lies in the fact that in spite of the thoroughness of the first operation—and obviously the operation was thoroughly done, because the old opening and the old sac remained entirely closed and perfect in all respects—within eight months the patient presented himself with a hernia apparently recurrent, but really new and the direct result of the original operation. If there is an inherent tendency in some of these cases to the production of a second hernia after the cure of the first, in the way I have indicated, you will see at once that it is practically impossible to devise any operation which will, with absolute certainty, cure an inguinal hernia so long as it is necessary to transfix the parts in bringing them together with sutures which are liable to split the tissues and provide a passage for potential herniæ.

The fact illustrated by this case is of great importance, for you will find it sometimes stated by those who have great confidence in the completeness of their methods that recurrences do not follow upon the methods which they adopt, and that if a hernia comes again in the same part in the same subject it is not a recurrence in the true sense, but a new hernia. The return of the hernia may be due to a recurrence, or it may be a new hernia, but the practical upshot is that a rupture has reappeared after the performance of the operation for the radical cure, and it matters nothing to the ordinary patient whether the hernia comes down again through the old opening which was closed by the operation, or whether the hernia comes through another opening. In either case the operation, from his point of view, cannot be regarded as successful. Another point of importance is this:—I believe it is impossible, I do not care what operation is performed or who the operator may be, to promise with certainty that patients who are subjected in their daily occupations to great strain and hard work will be radically cured of hernia in the sense that they understand the term, *i. e.* that instrumental support will be unnecessary, unless the operation has been performed early in life. If in this case I had in-

sisted on the patient wearing a truss, he would probably have been perfectly sound at the present time; but he preferred to do without a truss, and the result is as we have seen.

This is an instance, then, in which there has occurred a second hernia (a recurrence for all practical purposes) in consequence of the abdominal wall giving way near the site of the former operation.

Only a few days before operating on the case just described, I operated upon another man on account of recurrence of hernia after a radical cure. The original operation appeared to have been in some respects done well, inasmuch as the stitches were *in situ* and the parts held together; but the stitches did not appear to have been placed sufficiently far down, the result being that there was a true recurrence of hernia from the original operation being incomplete. In this second case the condition of affairs was thus:—the upper part of the inguinal canal and the opening for the original hernia were still closed by silk sutures which we saw *in situ*, but the lower portion of the canal had been left defective, probably for the cord, and the hernia had actually recurred in the true sense of the word, the sac having made its way through the lower part of the canal below the sutures used at the operation. This is an instance of the result which may follow what may be termed without injustice an inadequate operation. We do not know the condition of the parts at the operation, nor do we know what led to there being such a large extent of the inguinal canal left unclosed, as the operation was not performed in this country. If this patient, again, had worn a truss, this small hernia would probably not have recurred.

In the cases described the operation may be said to have been the cause of the reappearance of hernia, in the first in consequence of the splitting of the parts concerned, in the second in consequence of the operation being inadequate. There are a certain number of people in whom the parts are not, in my opinion, adapted for operative measures. The subjects I refer to especially are those in which the abdominal parietes are abnormally thin and weak—they are classified for the purposes of the public services as having "weak abdominal rings." The parts about the inguinal canal are thin and weak, bulging greatly on coughing, although no actual hernia can be said to exist.

The weakness in the majority of these patients is unfortunately not confined to the parts immediately about the rings ; a careful examination will show that the lower abdominal parietes are thin generally, and that there is a universal tendency to bulging of the lower belly, which inclines to become pendulous after the manner frequently associated with hernia in old people.

Subjects like these are, in my opinion, as a rule unfitted for the radical operation, unless it be distinctly made clear that the immediate improvement effected by the treatment may not improbably be temporary or only partial.

The tissues under these circumstances almost invariably "give" more or less unless a very sedentary life be led, and a reproduction of what is for practical purposes the original state is prone to follow. The ideal cases for the radical cure are those which occur in growing subjects (the younger the subject the more certain is the cure in the true sense to be), in whom the lower abdominal walls generally are not ostensibly weak ; the size of the hernia or its nature is not of great consequence so long as the subject is growing and the parts dealt with in the operation healthy, the natural developmental tendency being in such cases a great aid to bringing about a permanence in the changes wrought immediately by the operation. I made this statement some years ago, and increased experience leads me to persist in the opinion. Patients of this sort should, as a rule, if not always, be made quite independent of a truss of any kind by an adequate operation. In adult life the permanent cure is certainly less general if all classes of patients are considered. The question of permanence of the result depends, of course, a great deal upon the condition of the parts and the physique of the patient, but a factor of much more importance is the amount of strain thrown upon the parts subsequent to operation.

You will find, as I have already said—and it is natural that it should be so,—that recurrence after the radical cure is less often seen in the well-to-do than in the working classes, excepting those amongst the well-to-do who indulge in violent exercises.

I have often heard it stated as an evidence of the completeness of the cure in the well-to-do, that a patient is able to ride and shoot without recurrence, but the strain of riding (hunting for

example) in the ordinary way, or the rough walking sometimes involved in shooting, &c., is nothing to that to which a working man employed in heavy manual labour (a signalman or navvy, for instance) is subjected ; moreover riding, to which so much importance is often attached in this connection, is not in my opinion a severe test, for the position assumed in riding I do not regard as at all favorable to the descent of a hernia. Abduction of the lower limb is certainly not the position one would choose for reducing a hernia, and it can hardly be especially favorable to its descent.

I venture to impress this particular question of occupation upon you because I believe it to be of supreme importance in the consideration of the desirability or not of performing the radical cure. It is quite clear that an operation which in a clerk or other person leading a sedentary life would bring about a condition of things which may make him independent of instrumental support, might be quite inadequate in a person employed in heavy manual labour, or who is constantly subjected to excessive and violent strain from any cause. In the same way it is clear that parts which may be sufficiently strong to stand the strain naturally occurring in the case of the sedentary person, might be entirely unable to do so in the other type of individual I have mentioned. You must not suppose that I consider the operation for the radical cure of hernia a bad treatment because a recurrence may at times happen, or because a truss is not infrequently necessary in certain classes of subjects after operation ; on the contrary it is, in my opinion, an excellent treatment, but like most others it has its limitations. I am anxious that you should be aware of these limitations. Nothing brings so much discredit upon an operation as exaggeration on the part of the surgeon of the benefit probably obtainable from it ; and it is quite certain that no person who fails to recognise the limitations of the treatment we are discussing, is in a position to give sound advice upon the subject.

So far as private practice is concerned, you will, I think, find, unless my experience is exceptional, that the majority of patients who suffer from rupture seek surgical aid mainly with the object of getting rid of the necessity for using trusses. A certain number are led to seek advice by the regulations of the public services, and some are anxious lest danger should arise from the condition itself.

The principal object, however, of the majority is, as I have said, to be rendered independent of the use of instrumental support.

In advising on this subject let me impress upon you the importance, for the reasons I have already indicated, of a full consideration of the following points:

1. Age.
2. General physique.
3. Occupation and habits.
4. The condition of lower abdominal parietes.

The main object of the consideration being to determine whether, in the event of an operation being performed, the strength of the parts will probably be sufficient to meet the strain without the aid of instrumental support, which they may be called upon to bear in any given case.

With regard to the operation, I do not think the actual method is of great importance so long as it is properly carried out, and provided that its principle is sound. Every surgeon should, I think, be able to ascertain which operation produces in his hands the best result.

The more I see of surgery the more I am convinced that all surgeons do not perform the same operation equally well. I am also sure that because a given operation produces certain results in the hands of one surgeon, it by no means follows that the same results will accrue in the practice of the same method by another operator. I mention this because there is, I think, rather too strong a habit with some surgeons of speaking as if the radical cure of hernia could only be accomplished by one method of operation (*e. g.* Bassini's, Halstead's, MacEwen's, Kocher's, &c.), the method favoured naturally being that by which the individual surgeon has achieved the most success.

By a method the principle of which is sound, I mean one which includes (1) the removal and complete obliteration of the sac above the level of the internal ring, and (2) the complete closure of the hernial opening and restoration as far as possible of the inguinal valvular arrangements by suturing the conjoined tendon and internal oblique to Poupart's ligament. The latter purpose should be effected by a permanent suture, *e. g.* silk. In very young children and infants, however, a slowly absorbable suture, such as kangaroo tendon, usually suffices, in consequence of the developmental tendency of the canal to close, when the foreign body which the

hernia represents is prevented from descending through the canal after the sac lying between its margins has been removed:

Treatment of Deep Urethral Strictures by Internal Urethrotomy.—Dr. Gwilym Davis ('University Medical Magazine,' August), in a paper on this subject, says that the prevailing sentiment of the profession is against the employment of internal urethrotomy for deep strictures of the urethra—that is, for those involving the bulbous and membranous portions,—and he cites authorities in support of this statement. He compares internal with external urethrotomy, and expresses his belief that the comparative value of the two methods is slightly in favour of the internal method for effecting a cure.

"The ultimate ground," he says, "on which a decision must be reached is the relative dangers of the two procedures. There are two—hæmorrhage and sepsis, with or without urinary infiltration. As regards the former, I have heard of no deaths caused by it in internal urethrotomy, nor have I had any trouble from that source, but I have heard of a death from hæmorrhage after external urethrotomy, and have felt it necessary not only to most carefully ligate all possible bleeding points, but also to pack the wound snugly and retain the packing in place for several days. The question of urinary fever and sepsis is the real buttress on which the opposition to internal urethrotomy rests. The fear of this is largely a heritage from former practice rather than from recent results. It is practically eliminated by modern antiseptic methods. I am hardly prepared as yet to admit that urinary fever is due solely to an infectious element. Chills sometimes follow too closely on urethral manipulations to exclude other agencies. It is more likely to be due, at least to some extent, to nervous disturbance in addition to sepsis. Urinary infiltration is to be avoided by leaving a catheter in the bladder for three days, at which time the wound will have been so closed by plastic material as to prevent the urine gaining access to the tissue spaces. Sepsis is prevented by rendering the urine antiseptic, by administering antiseptics by the mouth as well as using them locally, and by observing a careful technique."

N. Y. Medical Journal.

CHAPTERS FROM THE TEACHING OF DR. G. V. POORE.

No. IX.

GENTLEMEN,—In speaking of poisons I shall limit myself very largely to poisons which have been used criminally. If I were to deal with every "poisonous thing" I should be trenching upon the domain of the pharmacologist, and I have no intention of doing that—at all events, not to a greater extent than I can help. If a poison is something which is absorbed, we must remember that it may be absorbed from many points. It is usually absorbed from the stomach, or let us say from the alimentary tract, but it may be absorbed from any other mucous membrane. Some poisons are absorbed from the skin, or from a mere scratch upon the skin. Poisons may be absorbed from the subcutaneous tissue. I need not dilate upon the use and abuse of the hypodermic syringe. Absorption from the unbroken skin is very limited in its range; comparatively few things are absorbed from the skin. It is doubtful to what extent the soluble salts are absorbed by it; some would say not at all. For instance, if sodium chloride were absorbed by the skin, it is tolerably certain that sea bathing would not be the pleasure that it is. We know that mercury is absorbed from the skin, and that mercurial inunctions are common. We know that the constitutional effects of belladonna are obtained when it is applied as an ointment; and it is possible that the greasy medium in which the medicament is incorporated helps its assimilation. But you may take it in a general way that absorption by the unbroken skin is rare. Poisons do not act until absorption takes place, and the more rapidly the poison is brought into contact with the blood-vessels by which it is absorbed, the quicker it acts. A dose of morphia acts far more quickly given hypodermically than when given by the mouth; and occasionally, when hypodermic injections were very much more common than they are now, when an injection was accidentally administered into a superficial vein the effect of the injection was almost instantaneous. Absorption is quickest when the poison is given directly into a vein, but it can hardly be called absorption then.

It is very rapid when given under the skin, and is rapid from the alimentary mucous membrane. Of course if you put say a dose of crude opium into the middle of a dumpling, and put some of that into the stomach with a big meal it will be a long time before the patient is under the influence of the poison. But if you put a solution of morphia into a comparatively empty stomach, the action of the poison is much more rapid. The action of a poison is rapid according to the area of absorption; there are poisons which act exceedingly rapidly, which presumably are absorbed from the pulmonary area as well as from the alimentary. I allude to such a substance as strong prussic acid. Chemically pure prussic acid is a thing which most of us do not know; it does not exist except as a chemical curiosity, but it has been said that people have been killed by smelling perfectly pure prussic acid, and there is no doubt it acts so quickly owing to its great volatility and to the large surface to which it is exposed for absorption. Again, absorption from a serous membrane is very quick, and experiments have shown that septic poisons particularly are absorbed with great rapidity from the peritoneum. The operative dose of a poison is the balance left between absorption and elimination. As soon as a poison is absorbed it begins to be eliminated, and your physiological lecturers have told you how very rapidly after injection into a vein certain salts appear in the urine; elimination commences at once, and mainly by the urinary tract. Elimination is so rapid that poisoning under certain circumstances does not take place. Amongst the most rapidly acting poisons are snake poisons. Take a virulent snake in an active condition, and use the poison from it, and the symptoms set in in a few minutes. It is a well-known fact that there is very little danger in sucking a snake wound if you have no cracks upon your lips. The explanation which is offered is that under those circumstances elimination by the urine takes place as quickly as absorption. So with regard to another organic poison, curara; that acts very much more vigorously when injected subcutaneously than when given by the stomach, elimination by the urine being rapid, so that there is not an operative dose circulating in the blood. The channels for the elimination of poisons are many. After a poison has been taken you can detect it in the saliva, for instance, or in the fæces, or in the urine, and

possibly there may be elimination of some poisons by the sweat. The latter I believe is only a possibility, not a proved fact. With regard to the alimentary tract as an eliminating channel, that perhaps requires a few words. When a poison such as arsenic is taken, it is very common indeed post mortem to find evidence of the irritation of the poison in the gullet, in the stomach, or in the duodenum and upper part of the small intestine. As a rule, the rest of the small intestine is free from symptoms of irritation, but sometimes those symptoms are evident again in the large intestine. I think there can be no doubt that the large intestine is a powerful eliminating organ. I call to your mind here that one of the symptoms of chronic mercurial poisoning is indistinguishable from dysentery. You get intense inflammation of the large intestine, dysentery is set up, mucus is discharged, and in this discharge from the large intestine mercury can be detected. That is the case also, I take it, with regard to many mineral and organic poisons.

Now when poisons are absorbed from the alimentary canal they are taken up by the portal circulation to the liver, and it is not surprising to anybody that a poison may have been absorbed from the alimentary tract, and yet may not be present in the alimentary tract, or may not be detected there, but may be detected in the liver, and when you make a post-mortem you must of all the viscera be most careful about the liver, because it has happened again and again that a poison such as arsenic has been detected in the liver when it has been got rid of by the alimentary tract, either by purging, vomiting, or natural absorbing, or by the three processes together.

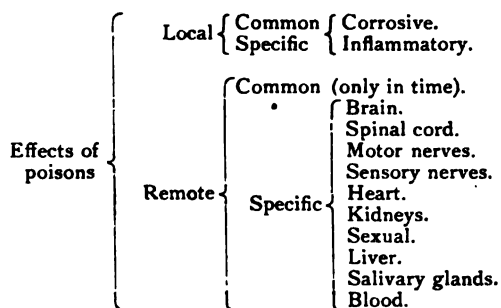
Then I take it that a poison may be eliminated not only from the large intestine, but also from any part of the alimentary mucous membrane. And it may happen, and has happened, that a poison given hypodermically has been detected in the stomach in small quantity. Therefore you will need to be on your guard about this.

Now it is obvious that the detection of small quantities of organic poisons in the tissues of the body is a chemical matter of very great delicacy and very great difficulty. And I should like to say that I do not think anybody ought to undertake a toxicological analysis for judicial purposes who is not a chemist; that is to say, nobody should under-

take it who does not make chemistry his profession and his first work in life. There are no more difficult problems than the detection of an alkaloid in a liver or in the tissues of the stomach, and such analyses have to be done with the greatest care; and unless a man is working all day and every day in the laboratory he ought not to undertake such work. In the old days, before the chemist's was a recognised profession, the medical man was the only person who knew any chemistry. But I remind you that the best that you can do, or the best that I can do, is not the best that can be done by a man whose principal work in life is in the chemical laboratory. But you medical men want to know something about the chemistry of poisons, because, perhaps, you are confronted with a case which arouses your suspicions as to poisoning, and you want to be able to see whether your suspicions are at all justified; you want to know how to be able to test fluids which the patient is taking; how to test vomit, urine, and so forth. These and other tests I shall hope to show you.

Now I will say a few words about the effects of poisons. Most poisons act upon many organs, and not only upon one. But it is nevertheless true that poisons have their seats of election for manifesting their action, and that poisons are taken up by the cells of one part more readily than they are by the cells of another part. You often hear it said that a certain poison acts, let us say, upon the spinal cord, and that another poison acts upon the nerve-endings in the muscles, and that it is eliminated by the saliva or urine; or that another poison acts upon the kidneys, and not upon other parts; that others act upon the uterus, and so forth. But I take it that the conclusion we must come to is that that is true, but true only in a limited sense. If I take a dose of curara I get the motor nerve-endings in my muscles paralysed, but when the end-plates of my motor nerves on the muscles have had their dose, it is tolerably certain, and may almost be conclusively shown, that other parts of the body and other cells then come in for their dose. I am inclined to say that certain cells in the body stand in relation to certain poisons just as a preference shareholder in a limited company to the dividend. The preference shareholder is a person who has the first draw upon the earnings of any commercial concern, and when the preference shareholder has had his dividend, then the holders of the deferred

shares get theirs. For instance, you may get bella-donna given you in only sufficient quantity to dilate the pupil, which is a preference shareholder; and when you get a bigger dose there is not only dilatation of the pupil, but delirium, quickening of the heart, and so on; and you get many parts of the body affected. Now it is very useful to have some kind of classification of poisons, but I should rather warn you that no classification of poisons I have ever seen is from every point of view satisfactory. I think it may help us in our grasp of the whole subject if I just draw up a scheme of the effects of poisons:



First of all the effects of poisons are of two kinds; they are *local* or they are *remote*. Let me explain what I mean. A person takes a dose of oxalic acid, which irritates the stomach and may cause perforation of it. That is a local effect. But after the local effect there is the remote effect, viz. great depression of the heart's action. Or, again, you may take carbolic acid. Carbolic acid is a violent irritant to the stomach, but after absorption it is a narcotic poison. With regard to the local effects of poisons, they may be corrosive or irritant. Then the local effects may not only be inflammatory but they may be specific, such as tingling of the lips and tongue produced by aconite, dilatation of the pupil by atropine, &c. With regard to the remote effects of poisons, these again may be classified. By common remote effect I mean the effect which is not specific, but is only remote in time. The best instance of that is stricture of the œsophagus following a dose of mineral acid. In specific remote effects we come to a list of the preference shareholders; for instance, take the brain. I suppose the brain is a preference shareholder in regard to alcohol. You take alcohol into the stomach, and it causes a warm sensation: that is a local effect; the specific

remote effect is delirium. Amongst the obvious spinal poisons is strychnia, which causes convulsions. Then we may have the motor nerves acted upon, and the best instance of this action is with curara, which acts on the motor nerve-endings, and possibly on the sensory nerves. Then there are some poisons which undoubtedly have a specific effect upon the heart, first upon the heart and then upon the blood-vessels. The best of those is digitalis, and the other so-called cardiac tonics. Then there are some which seem to act more upon the blood-vessels than upon the heart, and the best instance I can give you is ergot, which is a hæmostatic, but by no means exclusively. We know digitalis acts upon the vessels as well as upon the heart, and we know that ergot acts upon the heart as well as upon the vessels. Then there are some which act upon the kidneys, and produce violent irritation of those organs; and the best example I can give you is turpentine and bodies of that class. With regard to the sexual organs, that is rather more doubtful. Cantharides is said to cause priapism, and ergot and savin to cause contraction of the uterus. Ergot clearly must be classed among the bodies which act upon the uterus, but in regard to cantharides and savin it is very doubtful if the sexual stimulation which takes place is not merely a result of the violent irritation in the adjoining viscera.

Then with regard to the liver. Are there any poisons which act particularly upon the liver? A few years ago one would have said that mercury was one. Clearly phosphorus produces its most marked effects upon the liver. You must remember that almost everything goes to the liver. For the salivary glands, mercury and iodide of potassium seem to have a selective action. Lastly we come to the blood. The best example of a poison which acts directly upon the blood is carbon monoxide. This enters into combination with the hæmoglobin, and brings about death in that way by a positive act, and not merely by the negative act of depriving hæmoglobin of oxygen. I would repeat that though I have given you this table, no one is more conscious than I am of its shortcomings.

There are certain things which modify the action of poisons. For instance, nothing modifies the action of a poison more than quantity. As a rule the bigger the dose the more intense is the action,

but that is not an absolute rule. You may give a moderate dose of antimony and the person dies of cardiac depression; if you give a big dose he vomits the whole lot. The same with turpentine. If you give a big dose it sets up peristalsis, and the whole lot is voided as a purgative; but if you give a small dose urinary symptoms supervene. With mercury, again, the same thing holds good; if you give a big dose of mercury the patient is liable to be purged, and there are no later toxic symptoms. It is a fine line which divides toxicology from pharmacology, and you must not be surprised if I do not always keep to one side of the division.

Repeated small doses of a poison sometimes produce an accumulative effect. That is very much the case with lead. The next matter which influences the action of the poison is its form, the form in which it is given. A gaseous poison acts very quickly; such, for instance, as chloroform. A volatile fluid, again, like prussic acid, acts very quickly indeed. Then, as a rule, poisons act more quickly in solution than when given in the solid form. It goes without saying that the state of chemical combination affects the action of the substance on the body; for instance, strong sulphuric acid will poison, but some sulphates will not. A poison acts most quickly when it is given in the form of a solution into the stomach. A poison may not act because it is not dissolved. Poisons given in capsules may course through the alimentary tract without liberating the poison. Such a thing as a croton oil seed, if given whole, may go through the alimentary tract. The action of a poison is modified by the condition of the body. First of all as to habit, there is no doubt that we all of us do get habituated to certain poisons. The poison to which most of us get habituated is tobacco. We know that tobacco always makes a novice ill; he not only vomits, but he also gets severe cardiac depression, but that effect ceases to be produced in an incredibly short space of time. Opium is another poison which is very soon tolerated, and it is hardly too much to say that the opium-eater or the person who has the morphia craze cannot be killed by the drug. It seems as if they can take any amount. It is said that De Quincey took 333 grains of opium a day. Then we have to take note of such a thing as idiosyncrasy. Now, idio-

syncrasy pure and simple, apart from disease, I suppose we must accept as a fact. Some people are very sensitive to the effects of such things as opium, mercury, or arsenic. I know one lady intimately who is a great invalid, and has been seen by very many physicians. Not a few of those who have seen her have said she ought to have arsenic. I know for a fact, however, that it would be perfectly useless to try and give her arsenic. As little as $\frac{1}{4}$ minim of liquor arsenicalis will set up gastric irritation; she cannot tolerate even the smallest dose. You know there are patients in the hospital across the way who can tolerate one hundred times that dose. I take it that arsenic is one of those drugs which people do learn to tolerate, and there seems no doubt that in Styria, and in other parts of the earth, arsenic-eating is not uncommon. While on the subject of idiosyncrasy I would mention that I know very well a lady who twice in her life has been nearly killed by eating a Brazil nut. On the first occasion the act was followed in half an hour by an extreme nettlerash and swelling of the tongue, so much so as to threaten suffocation. Many years later she was telling her experience to some friends, and they said it must have been a bad nut. She then took the least little bit of another, and the same symptoms came on. There are certain articles of diet which some people cannot tolerate, honey being a common one. I need hardly say that disease makes a great deal of difference to the action of a poison, and the most important thing is the condition of the kidney. I have told you that the operative dose of a poison is the balance between what is absorbed and what is eliminated, and if the eliminating channels be blocked or in an unhealthy condition, the poisons will act very much more vigorously. It is very dangerous indeed to give certain poisons, such as mercury, to a man who is the subject of granular contracted kidney. I remember when I was clinical clerk there was a man under my care in the wards who had granular contracted kidney, and he had lost all his teeth. That happened in this way. He was in another hospital for some surgical trouble, and the man in the next bed was ordered a mercurial plaster for his testicle. By some mistake the mercurial plaster was put upon this man's testicle, and it brought on intense salivation, and he lost all his teeth. If you want to give mercury to a man with contracted granular kidney, give him a good

dose and follow it up with a saline purgative, and then it will very likely do considerable good. It is certain that alcohol, when given to a patient with acute fevers, does not have the same effect as when given to a man in health; it does not produce drunkenness or delirium, and if delirium is present it is usually diminished by the alcohol. Moreover, if you are giving only enough alcohol, and not too much, you do not get the alcoholic smell in the breath.

Now what are the symptoms of poisoning generally, and what circumstances should cause you to suspect poisoning in a patient? That is a very important matter. The effects of poisoning appear suddenly, and they usually follow eating or drinking or the taking of medicine. The acute effects of poisoning appear suddenly in health. There, again, we must be extremely careful. There are many diseases which appear suddenly, such as cholera. In a severe cholera epidemic a man may be well now, and dead in three hours; and during the great epidemics of 1854 and 1866 there were cases, even in this country, where the course was as rapid. Therefore do not rush to conclusions. You must remember that there is a condition which has often been mistaken for poisoning, namely, acute perforative ulcer of the stomach. Perforation of the stomach from this cause usually occurs after a meal, and some of these cases may simulate poisoning very closely indeed. When poisoning follows a meal we can often get some clue, whether it be accidental or criminal poisoning, by cross-examining everybody who was at the table, and ascertaining what the poisoned ones ate and what the non-poisoned ones did not eat. By these means you may sometimes be able to fix the channels through which the poison came. I need hardly remind you that sometimes food may be accidentally poisonous, and that such things as mushrooms and shellfish and pork may be in a very poisonous condition from what one may call accidental circumstances. Of course, when you suspect poisoning, that which clinches the diagnosis is the detection of poison in some article of food or in the excreta, and that is a matter which we shall have to give attention to when we deal with the poisons *seriatim*.

LATENT CANCER OF THE STOMACH.

BY.

JULIUS FRIEDENWALD, M.D., and A. S. HOTALING, M.D.

THE diagnosis of carcinoma of the stomach is at times very difficult. This difficulty is apparent in the 113 cases of carcinoma of the stomach collected by Guinard, in which the diagnosis had been made in only thirty-four cases. These cases, however, were collected from reports in the 'Bulletin de la Société Anatomique de Paris,' most of them being remarkable for some anatomical or clinical peculiarity in diagnosis. There are many cases of carcinoma of the stomach in which the cardinal symptoms are absent, and yet the diagnosis can be made from other symptoms; there are other cases, however, in which not only are the cardinal symptoms entirely absent, but all other symptoms, if present at all, are so insignificant that they do not lead one even to suspect a carcinoma. Such cases are known as latent cancer of the stomach.

There are two varieties of latent cancer—first, those cases in which the gastric symptoms are absent or so insignificant that they are masked by other general symptoms. Thus the carcinoma may be masked by some intercurrent disease, or secondary metastases from a primary gastric carcinoma may hide the primary disease. Secondly, those cases in which there are no symptoms whatever, general or local.

Of the first variety a large number of cases have been reported; of the second the cases are few. The pathological conditions leading to latency are that the growth be located on the anterior or the posterior wall of the stomach in such a position as to involve neither the cardiac nor the pyloric orifice, and that the surface of the tumour do not become ulcerated. When either of these orifices is involved symptoms of obstruction are at once manifested; if there is marked ulceration, toxic symptoms are produced.

Latent cancers of the stomach have been observed by Cruveilhier and Barth, and cases have been reported from the clinics of Trousseau, Andral, and Guéneau de Mussy. Chesnel has reported six cases; three of these were masked by pregnancy. In another case, in a man who had died suddenly from apoplexy, a cancer of the stomach was discovered at the autopsy. In still another case,

presenting the clinical symptoms of Bright's disease, a similar discovery was made.

Raymond reported a case in which there were no gastric symptoms whatever; there were a sudden and very high elevation of temperature and a general and apparently eczematous eruption. The true condition was revealed at the autopsy. In another case, that of a man aged 56 years, the only symptoms were marked cachexia and general oedema. The autopsy revealed a large carcinoma of the walls of the stomach, not involving the orifices.

E. Brissaud relates a case of a man aged 57 years, who entered the hospital suffering with intense anorexia; there was much emaciation, but neither vomiting nor any digestive disturbance. He continued in this condition for twenty-two days, when fever suddenly came on, with intense pain in the chest. A purulent pleurisy was discovered. Soon the joints became infected, and there were other manifestations of pyæmia. The patient died, and a large cancer of the lesser curvature of the stomach was discovered. Many of the joints were found filled with pus, and the left pleura contained a large quantity of pus.

Brodeur reports a case, that of a man aged 87 years, with a good antecedent history, who complained of loss of appetite, slight nausea, though not vomiting, and alternating constipation and diarrhoea. Oedema came on, and he died. At the autopsy a large encephaloid carcinoma of the stomach was found, not involving the orifices, but the greater curvature of the stomach was entirely destroyed, and its wall was replaced by the spleen; the left kidney and the liver were also involved. In spite of the extensive ulceration, there had never been vomiting or hæmatemesis or pain. This case is remarkable as showing how far the destruction of the stomach may go without producing any marked symptoms.

An interesting case is reported by Goldscheider. A woman aged 61 years presented symptoms of a compression myelitis, the cause of which could not be determined. At the autopsy a carcinoma of the spinal column was discovered, which was secondary to a carcinoma of the lesser curvature of the stomach. In this case there were no symptoms whatever of gastric disturbance.

Hampeln reports a number of cases. In one the diagnosis of senile marasmus and arterio-sclerosis was made; in another pulmonary tuberculosis; in

still another pernicious anæmia. In all of the cases carcinoma of the lesser curvature was found. In two febrile cases the diagnosis had been malaria and abscess of the liver.

Aron demonstrated before the Medical Association of Berlin a specimen of latent cancer of the stomach. The patient, a man aged 78 years, had become emaciated, and had oedema of the face and extremities, ascites, and fluid in the pleural cavities. Nothing could be determined by examination except the existence of an enlarged liver. At the post-mortem examination a large ulcerated carcinoma was found, involving the greater curvature of the stomach.

Leichtenstern reports three cases. In the first there was an acute compression of the spinal cord, with paraplegia, due to a secondary lesion in the stomach. This had not produced any gastric disturbance. In the second case the diagnosis of acute miliary tuberculosis had been made. There were ascites, abdominal pains, and symptoms of chronic peritonitis. At the autopsy miliary carcinoma of the peritoneum was discovered, secondary to a primary scirrhus carcinoma on the posterior wall and greater curvature of the stomach. The third case was one of chronic nephritis. There was hypertrophy, but there were no gastric symptoms except occasional vomiting. The trouble was believed to be uræmic. The patient died of uræmia, and at the autopsy, besides the cardiac and renal lesions, a carcinoma on the lesser curvature of the stomach was discovered.

Medical Record, Sept. 24th, 1898.

Eczema of the Lips caused by Mouth Washes and Tooth Powders was observed by Prof. WEISSER (Breslau) in a few cases. A boy, six years of age, was suffering for months from a squamous eczema surrounding the mouth. All treatment employed proved unsuccessful; but as soon as the mouth wash was discontinued the eczema at once disappeared. A similar experience was had in a second and a third case. In a fourth, which occurred in a young lady, the affection had existed for two years. After all mouth washes and tooth powders which contained olive oil or pepper-mint oil were relinquished a marked improvement of the eczema occurred.—*Wien. klin. Woch.*

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A CLINICAL LECTURE

Delivered at Guy's Hospital, October 8th, 1898, by

P. H. PYE-SMITH, M.D., F.R.S.,

Physician and Lecturer on Medicine at the Hospital.

GENTLEMEN,—To-day begins our clinical session of this winter, and I will bring before you an interesting case of cerebral disease lately in the hospital, which affords, by the death of the patient, an opportunity of verifying our diagnosis. I will preface what I have to say about the case by a word on the methods of diagnosis generally.

In the clinical lectures diagnosis and treatment are our subjects, and we leave questions of pathology, of the causation of diseases, and of prognosis to the systematic lectures.

Now in the art of diagnosis, that is to say the thorough understanding of all that can be learnt about a case of illness, what are our methods, and which of those methods are the most important? I suppose you will agree with me that we ought never to be satisfied with a diagnosis until we know not only the organ at fault, that is to say *where* the disease is, but also the nature of the disease, that is to say *what* it is—until we know the time it has existed, until we know what started it, what aggravated it, what checked it, and until we can form a judgment as to its subsequent progress. Then only can we be said to have made a thorough diagnosis. Such a diagnosis can be made in the case of scabies, where we know everything that can be known about the disease. The same is true of valvular disease of the heart, of pneumonia, of most of the exanthems, and of lead colic.

That, then, is our aim. What are our methods? I need only remind you of them.

First there is observation—studying the patient as we see him before us, and this study must, as you know, be prolonged and careful. We cannot tell what is the matter at a glance; you will find as you get experience in practice that as soon as the patient comes into your room you form an

involuntary guess as to what is wrong, and very often you will be right in your guess. Such cases are advanced phthisis, acne, tabes, and gin-drinkers' cirrhosis of the liver. But when you come to examine carefully you will sometimes find that appearances are deceitful. Therefore always reserve your diagnosis; never pretend to decide at once about any case that comes before you, but reserve your diagnosis until you have made a thorough examination. Sometimes, it is true, a patient is so ill that a complete examination cannot be made; you must at once relieve his pain or meet imminent danger. I would still say, make your diagnosis depend on your observation, but in that case let your observation be short; you must be content not to examine the whole chest, but ascertain the condition of the bases of the lungs behind and of their apices in front, and make out whether there is a murmur at the heart. Be content with passing your hand over the abdomen to feel whether there is ascites or an enlarged liver, or a pregnant uterus or other tumour. Note the characters of the pulse and breathing, and the heat or moisture of the skin; and as soon as possible get the urine to examine. You may then make a diagnosis accurate enough to treat your patient rationally, honestly, and with effect.

In all difficult cases, even after the first observation, write down your opinion, but do not say anything about it until you see the patient again. Often you will find that a case from which you went away puzzled, and which perhaps kept you awake at night, becomes clear when you examine it next morning in the daylight. On the second visit symptoms which were prominent and contradictory have disappeared. Other symptoms which you did not notice have come out clearly, and what seemed at first a very difficult case now becomes plain.

Again, suppose you have a case in which you have made your diagnosis, and have treated the patient in the way which you know is likely to do him good; but he does not improve, and gets rather worse than better. Then question your diagnosis, go back and begin the case again as if you had never seen the patient before. You will often find something which you had not noticed; you may find a new symptom, or one which you had previously overlooked.

What do we learn from observation? You know the distinction often made between symptoms and physical signs, but it is not one that holds. Every physical sign is a symptom, and many so-called symptoms are really physical signs. Again, if you examine the rectum and find a cancerous mass, will you call it a symptom or a sign, or will you not rather say that you have felt the disease itself, and do not want either sign or symptom? So if you use the laryngoscope and find a polypus in the larynx, that is neither sign nor symptom; it is the disease. The same holds good with regard to many affections of the skin. It is not worth your while to distinguish between signs and symptoms, but there are two distinctions which are worth while making: one is the distinction between what (if I may use a philosophical term) is subjective and what is objective. The subjective symptoms are those which depend upon the patient's own statement. A man says he suffers excruciating pain in his temple; another says that he feels so weak that he cannot move. Those are statements the truth of which you can check to some extent. If a man suffers from excruciating pain he cannot eat as usual; and usually a sharp pain will make the pupils dilate. Moreover, a man suffering severe pain cannot be distracted by conversation, but will still wear an aspect of suffering. Still in many cases you must depend almost entirely upon what the patient tells you. These never form so sure a foundation for diagnosis as objective symptoms, that is to say those which depend on your own sensations, which you can see or feel or hear, including those which you make out with the aid of the stethoscope, microscope, ophthalmoscope, laryngoscope, or thermometer.

Another real distinction is between the physical signs, which tell you only the physical state of an organ, the signs which tell you its physiological state, and those which tell you its pathological state. Many mistakes will be avoided if you keep clearly in mind that when you percuss a chest and find dulness, all that tells you is the physical state of the lung. If the proper pulmonary percussion note is not damped by dropsy, or fat, or thickened pleura, the dull note means solid lung. When a lung is resonant it contains air, and if you had that lung in your hand and put it into water it would float. But if you find dulness, that part of the

lung may be collapsed or filled up with exudation or new growth, but if you had it in your hand it would sink in water. Always connect resonance on percussion with contained air and floating in water, and associate dulness on percussion with absence of air and sinking in water. But remember that no dulness can tell you whether a patient has pneumonia; the dulness tells you that he has a solid lung, but the cause of the solid lung you must get at from other considerations. So that physical signs are the signs of physical conditions. If you feel a large liver or a large spleen, then the liver or spleen is large, but what makes it large you cannot tell by feeling.

Secondly, some symptoms and signs tell you of physiological conditions. The thermometer tells you that a patient is in a state we call fever, but it does not tell you the cause of the fever; the signs are just the same whether it is caused by inflammation or by direct neural disturbance; it tells you the physiological fact that the mean bodily temperature is increased.

The third kind of symptom is the pathological. The signs, however, are comparatively few. You learn from certain bacilli in the sputum that there is tuberculosis; as you learn from fatty casts in the urine that there is chronic tubal nephritis. So that we have three kinds of signs: (a) the physical signs, (b) the physiological signs, and (c) the pathological signs.

(2) So much for observation as a means of diagnosis. Next comes the *History*. Now, that is comparatively unimportant. Your natural tendency is to lay too much weight upon history. No doubt the thorough and complete account that one gets if one meets a practitioner who has had the patient under his eye from the time he brought him into the world and has known all his ailments, is of the greatest service, and that alone may be enough to base the diagnosis on. But such cases only occur occasionally in private practice, and never with hospital patients. People who come to you without your knowing anything about them will often give a history which not only does not help but misleads you. Again and again, without intention or wish to deceive, the history which you get from hospital patients is so confused, so inaccurate, so involved, lapses of memory are so frequent, and they so mix up what they know with what their friends have told them and what the doctor has

told them and what they suppose, that it needs the skill of an Old Bailey lawyer to get at the facts of the case. On the whole, you will economise time by examining the patient first and listening to his narrative afterwards.

Again, do not forget that probability is a treacherous foundation for diagnosis. In the case I am going to speak of, you will see that the probability was one thing and the fact another. Do not jump at the conclusion that because a man is a plumber and has pain in the belly he has lead colic; he may have strangulated hernia; or that because a man has syphilis and also has paralysis, that the paralysis is due to a gumma. Syphilis does not protect from cerebral hæmorrhage any more than from scabies, or psoriasis, or scarlet fever.

(3) There is a third method of diagnosis beside those I have mentioned, and it is one which you are familiar with, though perhaps you have not looked at it in this light, and that is knowledge of pathology. There is an old saying, and a very true one, "the road to an accurate diagnosis is through the deadhouse." The meaning is that unless you have a thorough knowledge of the things which are possible or probable in the way of disease, you will never be able to make out obscure cases. You must know that the liver is liable to certain diseases, and none other; that the brain is liable to certain diseases, and none other; and so on. Otherwise, if every organ of the body had every disease of which the body is capable, we should never come to a diagnosis, and the patient would either have recovered or died before we found out what was the matter with him. But we know that if we ascertain that the patient has, say, a large liver, there are only a few causes of this enlargement, half a dozen at the most, which you have to think of. So if you make up your mind that a patient has a tumour of the brain, you know that most of the tumours which occur in the body generally are not found in the brain, and these you need not consider. Again, some of those which may occur in the brain are very rare; you meet with them in museums, but they are only interesting as curiosities. I have seen cysticercus of the brain twice only in my lifetime. Just think of the difference in frequency between a cysticercus and a tuberculous tumour of the brain. Probably you have all

seen a tuberculous tumour of the brain, one of the commonest cerebral tumours, and by far the commonest in young people. But cysticercus is a curiosity, and if I were to see a case of cysticercus of the brain to-morrow I should not diagnose it, because its effects are like those of any other tumour. When we find such and such possible lesions of an organ we decide between them by pathological probability. If you find a lump in the liver, it is far more likely to be carcinoma than anything else in the world. If you find a fluctuating tumour of the liver, it is almost certain to be hydatid or an abscess.

Thus your knowledge of pathology will often guide you in diagnosis on this principle; that things which are very rare may be dismissed, and that those which are most common are most likely.

Now we come to the particular case that I am going to speak to you about. It is that of a girl aged 16, previously healthy, whose family history is very properly set out here; but neither it nor the previous history of the patient have any bearing on the case. She was apparently a healthy girl until the 24th of last month, and she died on the 5th of this, that is to say eleven days after her illness began. On September the 24th, when apparently well, she went to the funeral of a relative, and, as the mother said afterwards, "she seemed very much upset." That is a history which we disregard. We do not know any disease which is caused by attending a funeral; we do not even know of any disease which is caused by grief. You know that the pages of history contain numerous examples of people having died of a broken heart; but if so it was not from grief. The heart breaks either from injury of the right ventricle or from disease of the walls of the left. George II died of a broken heart, but it was not from grief. In this girl's history the funeral was a mere accident. People always want to find a cause for disease. No doubt the true cause is a most important thing to find, but in order to find it you must beware of accepting such explanations as overwork, grief, "worry," exposure to the weather, and so on. Even such real causes of disease as alcohol must be proved, not assumed, for although alcohol causes much mischief its effects are limited and definite. It is absurd to hold alcohol or syphilis

or malaria responsible for every disease that may follow it.

This girl came home from the funeral and had a fit. The fit consisted in convulsions of which we have no exact account. After the fit she complained of severe headache, and was brought to the hospital. She could just walk into the ward, though very weak, and she could speak, though somewhat indistinctly. She had not aphasia; the indistinct speech was due to partial paralysis of the left side of her face, and she had also partial paralysis of the left arm and leg, that is to say left hemiplegia affecting the face on the same side as the limbs. The hemiplegia gradually disappeared, or very nearly disappeared, and her speech became as usual. She seemed to be getting better; she could feed herself, and for three or four days was convalescent. Then on the 30th of September, a week after she was admitted, she had another fit early in the morning. This was watched more carefully, as it was seen by skilled observers. It began with vomiting, then both her arms became stiff and rigid, and she again lost consciousness, though not completely. Next the arm and leg began to jerk, her tongue was bitten, there was nystagmus, and the pupils were noticed to be equal and dilated. This fit lasted five hours. It was epileptiform, but most of you will already have seen that it was not an ordinary epileptic convulsion. In the first place, it lasted too long. Sometimes an epileptic fit only lasts five minutes, sometimes not more than five seconds, and never more than a quarter of an hour. Therefore a fit of five hours cannot be a manifestation of true epilepsy. Then, again, she did not quite lose consciousness. An epileptic fit, however slight it may be, even *petit mal* which lasts a fraction of a minute, is always accompanied by loss of consciousness. Again, there was not only jerking of the limbs, that is clonic convulsion, but also rigidity, that is tonic contraction; and afterwards there was weakness in the affected limbs. This points not to ordinary epilepsy, but to what is sometimes called (after Dr. Hughlings Jackson), Jacksonian epilepsy—not a mere functional disturbance, but due to some organic change in the brain. Eclampsia is a convenient word to use for a convulsive fit, and I think it is well we should only speak of true "epilepsy" when the fits are not secondary. This was a fit which

pointed to organic disease of the brain. The hemiplegia again to some extent cleared up, as it did after the first fit, but for three days there was incontinence of urine. Now that is a very important sign. Scarcely ever will you find adults, and I think you will never find women, who fail to hold their water at night except against their will. Hysterical girls retain their urine; they do not pass it in bed; so that incontinence of urine is always a serious symptom; it is not due to a functional disturbance.

The next symptom was that the temperature went up, and it rose very suddenly and rapidly, as this chart shows, namely to 104° and subsequently to 105.8° ; it was a very high temperature and very irregular. This symptom bore one of two interpretations, which were both discussed at the bedside. A common cause of high temperature is inflammation, and on the whole I thought it was probably the case here, and that she had acute meningitis. But we must not forget that lesions of certain parts of the brain will cause a rise of temperature quite independently of inflammation or sepsis. The fact has been observed for many years, but of late much more attention has been paid to it. If you look in the forty-second volume of our 'Reports,' about twelve years ago, you will see an interesting paper by Dr. Hale White in which he collects a considerable number of cases in our wards, and refers to others observed abroad. I remember a patient of my own, a boy of eleven, who died from tumour of the brain without meningitis, after high temperature during life. The corpus striatum contains thermotaxic centres, but there are probably others.

Our patient became steadily worse from this time. There had been some difficulty in examining the fundus of the eyes because of the nystagmus and restless movements of the head, but obvious and severe optic neuritis was clearly seen in one eye. In spite of cold sponging the temperature went up again and again, she became deeply comatose, and the respirations rose to 80 per minute, the pulse became imperceptible, and she died early on the morning of the 5th inst.

That is the case, now for the diagnosis. It is a good plan to start from some prominent objective symptom. If you feel a large liver, think of the causes of enlargement of that organ. If you hear a cardiac murmur, think of the various causes of

disease of the heart. If you see hæmoptysis, remember the causes of blood spitting. This was obviously a nervous case. The paralysis, the convulsions, and the optic neuritis, the temperature and the final coma, all pointed to cerebral disease.

Always make a diagnosis of some sort, never say you cannot make anything of a case; even if you only say dropsy, or something wrong with the liver, it is better than saying nothing.

When you have to deal with a nervous case, think in the first place of the possibility of shamming. It is a rare occurrence, but such cases do happen, and if it occurs, and you do not happen to think of it, you may not only fail to cure your patient, but you may damage your own reputation. If you are surgeon to a jail or a reformatory, you must not be surprised to have cases of feigned disease or injury. If you are the medical officer of a public school you have to remember the possibility of shamming; and one is ashamed to say, that if you enter the services you must not shut your eyes to the possibility of a soldier or a sailor pretending to be ill when he is well, and you are bound to recognise such cases. It is not difficult, by the exercise of your common sense and experience, to find out malingerers if you only think of them. The chance of your being deceived is that you may not always think of the possibility of shamming, just as you may forget in cases of gastritis to think of poison, or in cases of abdominal tumour to think of pregnancy.

Next comes hysteria, which is a very different thing. You may meet with a shamming woman, but the majority of shamners are men and boys. You may meet with hysteria in the male, and among boys it is not uncommon. At the same time the enormous majority of hysterical patients are women. It is true that hysterical women may mangle, but most of them are anxious to get well; their ailment is a defect of will, not of intention, and it would be grossly unfair if you were to accuse an hysterical girl of wilfully pretending to be ill. That is not the case, and you help them all the more if while recognising that there is nothing dangerous the matter you recognise also that they are suffering from a distressing form of nervous malady which is not entirely under their control.

Next comes the question, is the disorder functional, such as epilepsy, of which I have just spoken. By functional we mean that if you made a

post-mortem examination on a patient the subject of functional disease immediately after death, you would find nothing wrong, either with the naked eye, with the microscope, or with all the methods of hardening and staining that we have now at hand; neither would a bacteriological examination reveal anything amiss; you would find absolutely nothing to distinguish it from the brain or nervous system of a healthy man.

There is still another form of nervous disease you must not forget, the toxic neuroses. In such cases you find no anatomical change post mortem, but you can find a chemical change; and we know that they depend upon poisoning, either with a metal like lead, or with a plant like belladonna, or with an animal poison like a toxin or an albumose.

When, lastly, you come to structural diseases, think first of traumatic affections. If a man is brought comatose into the surgery, first think of fractured skull, next of drink, and thirdly of opium. But here, again, do not forget the possibility of shamming or of hysteria. It is only when you can put all these things on one side, that you can decide a case to be one of organic disease. In the case I am speaking of we had no difficulty in arriving at a diagnosis of structural disease. The patient was a girl, but was not one of those who pretend. She had no symptoms of hysteria. Hemiplegia is never toxic, and in her it was of the ordinary kind, partial and chiefly motor, not complete and anæsthetic as it is in functional cases. Optic neuritis cannot be feigned, and the fits from which she suffered were neither hysterical nor idiopathic epilepsy.

The diagnosis so far was manifest, and it was only when one turned to the inevitably fatal prognosis that one felt a faint hope that the case might, after all, be one of those strange instances of recovery from apparently hopeless nervous disease that we occasionally meet in women.

There is a power in the female organism which produces symptoms of organic disease, and yet admits in rare cases of recovery, unforeseen and inexplicable.

When we had decided on the presence of structural disease in the nervous system, we had next to determine its seat and its nature. Consider the muscles as end-organs of the motor nerves, for paralysis may begin in the muscles, like the pseudo-

hypertrophic paralysis of Duchenne. Do not separate such myopathic palsy from other atrophic paralyses, for clinically they are closely allied. Next look to the peripheral nerves, the seat of most toxic paralyses—diphtherial, alcoholic, and plumbic. In the third place comes the cord, with its prolongation into the skull, *medulla spinalis oblongata*, or, as we now call it, the bulb; and lastly, the brain. In the present case the symptoms were cerebral. You are all aware that hemiplegia points to a cerebral lesion, while paraplegia means disease of the cord. To localise our diagnosis further, was the disease in the meninges or in the cortex, or in the internal capsule? Was it in the frontal or occipital region, in the cerebellum, or in the pons? Clearly it was in the motor tract of the right side; not in the bulb, not in the crura, perhaps in the corpus striatum, and probably, as I thought from the character of the eclampsia, in the motor tract of the cortex. When you get as far as that I advise you to stop and ask another question, not where is the lesion, but what is its nature. It might have been hæmorrhage; that is a common cause of hemiplegia, but that does not cause fits nor optic neuritis. You may add that a girl of sixteen years of age is not likely to have cerebral hæmorrhage, but such cases do occur.

Embolism is another lesion it might have been. But an embolus is a detached thrombus. If so, where did it come from? From the left side of the heart? That means either fungating endocarditis or else mitral narrowing and a clot forming in the left auricle. But here there was no murmur, no distended auricle, no likelihood of embolism. Then there is red softening as a primary condition, very rare, and almost unknown in young people. Cerebral thrombosis, again, is unknown at our patient's age, and when it occurs in the sinuses of infants has very different characters. As to abscess, there again pathology helps us. Cerebral abscess is never primary; it is always the result of injury or of local or general pyæmia; most often, as you know, due to caries of the ear or nose or some other part of the skull, sometimes to suppuration in the chest or elsewhere. But abscess of the brain as a primary condition in a previously healthy person does not occur. Therefore we may exclude it, though it might account for the symptoms of our case. Then we come

back to meningitis. I confess I thought this condition was present, because she was at the age for it, because the temperature was high, the course very rapid, and because vomiting, convulsions, optic neuritis, and ingravescent coma very often accompany meningitis. But there was more than meningitis; that may cause optic neuritis, but it could scarcely cause these curious fits, and therefore it seemed probable there was some lump, some focal lesion in addition to meningitis. What would it be likely to be? What is the commonest tumour of the brain in children and young people under twenty? Far and away the commonest tumour is tubercle, and next comes glioma. Assuming, then, that the lesion was a tuberculous meningitis, with a tumour of the same kind, we turn back to the local diagnosis and fix the former in the base and the latter in the motor tract of the right hemisphere, perhaps the cortex.

The objections to this diagnosis were the absence of ocular paralyses which usually go with basal meningitis, and the absence of signs of tubercle in the lungs, abdomen, bones, and joints. Still, if we never make a diagnosis till we have every symptom in its favour, we shall in all but the simplest cases never make one at all.

Now comes the post-mortem examination. My diagnosis turned out to be wrong. Not absolutely wrong, for there was organic disease found, and it was situated in the brain in the right hemisphere, and in the motor tract. So far we had arrived about as near as an eminent predecessor of mine who flourished here early in the century. He lived long before the discoveries of Laennec, of Bright, and of Helmholtz, but it was said of him that when he had placed the seat of disease in one of the three great cavities—the skull, the chest, or the abdomen—the post-mortem examination never showed it to be in another. That was a modest degree of accuracy, but to my mind it shows far more acumen for him to get as near as that, than for us to get as near as we sometimes do now. There was no meningitis, and the tumour was not tuberculous. Neither was it what is the next in frequency—glioma. It was a cyst. Now cysts occur in the brain only now and then. Some of them are parasitic, cysticercus or echinococcus, but they occur very rarely. Others are, no doubt, the result of hæmorrhage from which the blood is gradually absorbed, leaving only the serum behind.

That is very unlikely here, because hæmorrhage is rare at sixteen, and there was no pigment or hæmin crystals left behind. It was not parasitic, it was not hæmorrhage, but it may have been a glioma which underwent cystic degeneration, and I am inclined to think it was so. The cyst wall has not yet been examined microscopically, but we may find some little bit of soft gliomatous tissue which would prove that to be the case. If not, we must call it a simple cerebral cyst. Such cysts are not very uncommon in cerebrum or cerebellum. Usually they are solitary, as in this case, but I have seen them multiple and associated with cysts of the liver and kidneys. Clinically, as we might expect beforehand, there is no difference in the symptoms of such a single large cyst and a solid tumour in the same spot; they both act alike as foreign bodies. If a leaden bullet had been put into this girl's skull in the place where the cyst was, it would have produced exactly the same effects. Therefore we diagnose the different kinds of tumour, not from symptoms, but from our knowledge of pathology.

Now when a patient dies there are two or three questions we should always ask ourselves; I mean after the post-mortem examination. If we do not hold a post-mortem we learn nothing and gain nothing from the case, which may help ourselves and others in the future. It is all unmixed failure and regret. Therefore let me urge you to make an autopsy whenever possible; nothing shows the thorough practitioner so much as his eagerness for this thankless but most interesting and most important duty. Every hour you spend in the post-mortem room while you are here will be time well spent, and will be invaluable in future practice.

The first question we ask is, was our diagnosis right? I have drawn on the board what we did find. This is a horizontal section through the right ventricle, showing its anterior horn, the cauda, and the lenticulus, the thalamus, and the internal capsule, with its genu. It was in the posterior limb of the internal capsule that the cyst was found, about the size of a French olive. It encroached slightly on the lenticulus and rather more on the thalamus. Its situation explained the hemiplegia, and the other symptoms were due to the fact of a tumour being in the cranium, interfering with cerebral circulation and nutrition.

Well, our diagnosis was not accurate at all. I

thought there was meningitis, but there was not. Perhaps more important than that, I thought it was a tuberculous tumour, when it turned out to be a cyst.

Secondly, we ought to ask ourselves, was our prognosis right? Did we expect the fatal termination? Here the prognosis was obvious. The poor girl had organic disease of the brain, and as she had incontinence of urine and fæces, and increasing coma, a very rapid pulse and very high temperature, there was clearly but a short step between her and death.

The third question, the most important of all, did we do all that could be done to save her? Yes, so far as our present knowledge goes, because in such a case we can do nothing. If we had diagnosed accurately the seat and nature of the tumour, you can imagine the possibility of the surgeon reaching the cyst and removing the contents. But I am not at all sure that even then the event would have been different, and at present I am afraid we are very far from any such accuracy in diagnosis.

Those are the questions which we ought to ask after a post-mortem examination; and I hope you will agree with me that although we always learn something from a case in which we are right, we may learn still more from a case in which we are wrong.

Hypodermic Injections of Alcohol and Tincture of Belladonna in the Treatment of Apparent Death in the New-born.—According to the *Semaine Médicale*, Dr. Brown, to the classical means of combating apparent death in the new-born, has added a new remedy. He is accustomed to inject under each arm of the child five drops of whisky, to which has been added a drop of tincture of belladonna. Soon after the child opens its eyes and utters a cry, which is indicative of the beginning of respiratory function.

In cases where alcohol and belladonna do not resuscitate the child immediately, the author gives a hypodermic injection of from four to eight grammes of hot sterilised water, and introduces into the stomach the same quantity of this liquid, to which has been added a drop of aromatic spirits of ammonia.—*St. Louis Medical and Surgical Journal*, October, 1898.

CLINICAL LECTURES

ON

MUSCULAR TREMOR AND CLONIC SPASM.

Delivered at the Hospital for Diseases of the Nervous System, Welbeck Street, by

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Physician to the Hospital.

LECTURE I.

Cases of Chorea, Rhythmical Chorea, and other Choreiform Movements.

GENTLEMEN,—The subject of muscular tremor or clonic spasm is one of much interest, and the symptom is one of considerable frequency in disorders of the nervous system. Although in a good many instances the anatomical lesion has been identified, there still exists a large proportion of the class we call functional, and especially of that large non-descript group, hysteria. We have quite a number of these cases attending the clinique just now, and I propose to take the opportunity of studying them with you.

The other day, in the out-patient room, a tall girl of 15, who had never menstruated, but had had no previous illnesses of any consequence, applied for what appeared to be a mild attack of chorea, almost confined to the right side of the body, and very much worse in the right arm. To the casual observer the movements had that disorderly character so typical of chorea, the age of the patient was quite consistent with that disorder, and there seemed to be no reasonable doubt about the matter. But on making her carry a spoon to her mouth or perform any other voluntary muscular action the oscillations completely ceased. Now you know that in the movements of chorea there is nothing more characteristic than the feature that the movements become greater as the muscular action proceeds, and in using a spoon it very often misses its goal.

I let her sit down opposite to me while I continued my work, and then, unobserved by her, I noticed two other important features: first, that although the movements were almost continuous,

there were paroxysms of greater severity from time to time; and secondly, that the irregularity of movement was only apparent. They had, in fact, a regular irregularity about them which occurs in hysterical movements, and often gives rise to much difference of opinion amongst observers. I then found that she came of an hysterical stock, and that the tremors had supervened somewhat suddenly seven weeks before, after an "upset" at home; and, moreover, that she herself presented the "ovarian" phenomenon. The case, in short, was one of hysterical choreiform spasms, and belonged to a group of diseases which is not inaptly called rhythmical chorea. She was treated by tincture of valerian and ammonium bromide, and made a rapid recovery.

This case will serve us as an excellent text for our investigations to-day, for it illustrates two important points. The first is the great difficulty in the diagnosis of choreiform movements and other clonic muscular spasms. The second is the importance of not being satisfied with a cursory glance at such cases, for in many instances it is only by repeated and prolonged observation, under a variety of conditions, during rest and during action, that a correct conclusion can be arrived at.

Before passing to the other cases I should like to say a few words about clonic spasm and tremors in general. Increased muscular action may, as you are aware, be of two kinds: it may be intermittent, when it is called clonic spasm; or it may be continuous, when it is described as tonic spasm. And it is the former, the intermittent or clonic muscular spasm, which constitutes the subject of our studies to-day. When the movements are small they are spoken of as "tremors," when large they are known as "clonic spasms." In either case they result from an alternating contraction and relaxation of the affected muscles, and must not be confused with the inco-ordination, *i.e.* the exaggerated or uncontrolled movements of tabes dorsalis, although that disease is sometimes included in this group. Nor must these tremors be confused clinically with the unsteadiness of movement which arises from weakness of the muscles, such as that which is sometimes associated with cerebellar disease.

Nevertheless, I hope to be able to prove to you that muscular tremor is clearly an evidence of incipient destruction—a destruction which is partial

and incomplete in its nature—of the neuro-muscular apparatus. Irritative lesions produce either *tonic rigidity* or *convulsions*. Destructive lesions produce *paralysis*. But when the destructive process is gradual, slow, and incomplete—when the lesion is not irritative enough to produce rigidity or convulsions, nor destructive enough to produce paralysis by completely destroying or cutting off all motor nerve-impulses—then do we get *tremor* and unsteadiness of movement, just as a candle flame flickers in its socket before it finally goes out. At the present time this is no mere theory; there is abundant clinical evidence, as we shall presently see, to prove that *incomplete destruction*, or what amounts to the same thing, *partial recovery*, produces muscular tremor or clonic spasm of the muscles supplied by the diseased nerve structures. I shall refer to this matter again, but it will be well to keep it constantly before our minds in studying the clinical features of muscular tremor and clonic spasm.

Here is a patient, E. M—, æt. 73, and you will observe, as he walks along, the tremulous uncertainty and irregularity of his voluntary movements. They are the clinical evidences of the incipient degeneration or destruction of his nerve structures.

But our chief concern this afternoon is to discuss the differentiation, prognosis, and treatment of the various conditions which may give rise to muscular tremor or clonic spasm. Now the differentiation of these maladies forms one of the most difficult parts of neurology, and I have therefore ventured to submit to you a table arranged on a clinical basis, presenting a fairly complete list of these affections. I have arranged them in two groups: (a) CLONIC SPASMS, in which the movements are *large* and for the most part *irregular*, of which chorea is the most frequent and the most typical example; and (b) TREMORS, or movements which are *small* and usually *regular*, of which paralysis agitans is the type.

This table shows at a glance the different disorders with which we shall have to deal, and thanks to the resources of this hospital I shall hope to show you several very interesting and remarkable examples. But it must be remembered that although I have arranged them in this way, the same disease may belong at one time to one group, at another time to the other. Thus you will ob-

CLINICAL CLASSIFICATION OF TREMORS AND CLONIC SPASM.

CLONIC SPASMS.	TREMORS.
In which the movements are larger and often irregular (of which chorea is the type).	In which the movements are small and more or less regular (of which paralysis agitans is the type).
I. Chorea.	I. Paralysis agitans.
II. Hysterical or Rhythmical chorea (chorée rythmée) and its various forms, viz. dancing chorea, hammering chorea, &c.	II. Senile tremor.
III. Post-paralytic clonic spasm (descending sclerosis).	III. Disseminated sclerosis.
IV. Athetosis.	IV. Hysterical trembling.
V. Myoclonus multiplex.	V. General paralysis of the insane.
VI. Habit spasm.	VI. Alcoholic tremor.
VII. Clonic facial spasm.	VII. Metallic tremor.
VIII. Spasmodic torticollis.	VIII. Other toxic conditions.
IX. Fatigue spasm.	IX. Nervous or neurosenic tremor, and occupation neurosis.
	X. Other diseases in which the tremor is not always a prominent feature, viz. Graves' or Basedow's disease.
	Amyotrophic paralysis.
	Friedreich's disease.
	Tumour of the brain under certain conditions.
	Lateral sclerosis (later stage).

serve that I have placed hysterical movements in both lists; and although the movements in disseminated sclerosis are usually small, they may be large and irregular.

Clinical investigation.—We have to do here with an increase of muscular action, an increase which is intermittent; and the points to note about such cases are, first, the *distribution* of the tremor or spasm. In certain cases of hysterical tremor the movements may be localised, and so also in torticollis, habit-spasm, and facial spasms. In paralytic dementia, also, it is at first almost confined to the lips and arms, but the distribution in most of the cases of muscular tremor is general, or soon tends to become so. The *rate of oscillation* is another feature worth observing. By some it is regarded of sufficient importance for a primary classification.* In paralysis agitans, senile tremor, and disseminated sclerosis the movements are usually slow, averaging 250 to 300 per minute; while in alco-

holic and metallic tremors, general paralysis, and Graves' disease they average 500 or 600 per minute. But these rates are very inconstant, and depend very largely on the circumstances of the moment. The *size* or *range* of the movements is, I believe, a more important point, and on this feature, combined with the next, I have based our primary division, which answers fairly well excepting in the case of hysterical movements. The *rhythm* is another important point to be noted, and it will often afford us valuable aid, for it so happens that the tremors or clonic spasms of small range are generally regular, whereas the larger ones are frequently irregular in rhythm. Thus the tremors of paralysis agitans, alcoholism, paralytic dementia, and others in the second group are characteristically regular, whereas the choreiform movements in our first group are characteristically irregular.

Special inquiry should always be made as to whether the *tremor is dependent upon voluntary muscular action or not*. It is convenient to have a single word for the description of this characteristic, and the Germans have adopted the word "Intention-zittern" for tremors which are only present during muscular action, and it is customary in England to speak of these as "intention tremors" or "voluntary spasms." Personally I prefer the term intention tremor, because the other implies that tremor is or can be controlled by the will, which is incorrect. One has to bear in mind that almost without exception all tremors and spasms are increased by emotion, and to some extent by voluntary muscular effort. But in the early stages of disseminated sclerosis, lateral sclerosis, metallic and alcoholic tremor, post-hemiplegic tremor, and Friedreich's disease the oscillations only begin when the patient makes a voluntary muscular movement. On the other hand, the tremor of paralysis agitans, senile tremor, and chorea are independent of any voluntary action on the part of the patient. Hysterical movements occupy almost a unique position in this respect, for in most cases, if they are influenced by voluntary muscular action at all, they cease or become less, as far as my observation goes, at any rate in cases of moderate severity. Other points to be observed are the *mode of advent*, the *course* which the disease runs, and the *age of the patient*. I have ventured to dwell on these details on account of the difficulties which arise in the diagnosis of these cases.

* Charcot, 'Leçons cliniques des Maladies du Système Nerveux,' vol. iii.

1. *St. Vitus' dance*—the chorea described by Sydenham—is the most frequent and typical form of this kind of muscular disorder. There is no doubt that many of the conditions we are about to study were formerly regarded as aberrant forms of this disease. Even in the present day the entire group of clonic spasms is sometimes described under the term “choreiform movements.” Unfortunately many of them have come to be called “chorea” with an adjectival prefix, *e.g.* hysterical chorea, post-hemiplegic chorea, &c.

The most important characteristic of the movements in true chorea—the disease which is met with almost solely in children between the ages of five and fifteen, though I have seen undoubted cases at the ages of three and at thirty-nine—is their *irregularity* both in rate and range. They have no kind of rhythm or cadence, and though voluntary acts increase the jerkings, they come on quite independently of these. That the movements should cease when a voluntary action is commenced is wholly unlike any kind of true *St. Vitus' dance* I have ever seen, though I have seen it very frequently in the choreiform movements of hysteria. It was, you will remember, this feature which first made me doubt the diagnosis of chorea in the girl whom I showed you at the outset.

The oscillations of chorea convert the voluntary movements into gesticulations; and you will observe in the little boy who is now coming into the theatre that as he attempts to bring the spoon to his mouth he misses the goal. In disseminated sclerosis, on the contrary, the movements only come on when the patient begins a voluntary action; and although they may increase during muscular action, the patient nevertheless is able to insert the spoon in his mouth without difficulty. The clonic spasms of chorea may involve any and every muscle in the body—the face, the tongue, the muscles of deglutition, the diaphragm, and even the respiratory muscles. Sometimes they have a tendency to be one-sided; sometimes, as in the patient before you, they predominate in one limb. Not infrequently there is some muscular weakness in the affected limbs; and nearly half of the cases are complicated by cardiac valvular disease, which is wholly indistinguishable from that which complicates acute rheumatism. The disease runs a definite and generally benign course of several weeks, having a

spontaneous tendency towards recovery. It arises usually in nervous children, and is said to be sometimes spread among children by “imitation.” I have myself observed it to affect several of the children in a ward after the admission of a single case, and I would ask you to observe that all the conditions of this apparent “imitation” are capable of explanation by “infection.” For my own part, I believe that the true pathology of chorea will one day be written in the annals of bacteriology. I shall hope to set before you other reasons for this conclusion, but I must now ask your attention to the child before us.

He is a nervous little chap, aged 8. His nervousness was manifest some six weeks ago, when his mother sent him shopping with a shilling. Unfortunately he lost the shilling, and when he returned home his mother says he was “as white as a sheet,” and trembled so much that he could not stand, though his parents are not particularly strict nor dreaded by him. Last Thursday week (ten days ago) his teacher struck him over the right upper arm with a ruler for a fault which, of course, he says was that of another boy. The next day his father spoke to him about “fidgeting so much at the dinner-table,” but it was found that he could not help it. This was the beginning of the disease. The movements, you will observe, are very much more marked in the right upper extremity—the one which was struck. Elsewhere they are only occasionally observed. You know well that an attack of gout and the seat of the gouty manifestation may be determined by an injury. We know also that in hysterical manifestations the locality may be determined by the seat of an injury; several such cases have been recorded by Charcot.* Here, also, is a similar circumstance as regards chorea, a fact which has not, I believe, been hitherto observed in connection with this disease. This fact does not shake my belief in the bacterial origin, inasmuch as the manifestations of gout, another blood-disorder, and their locality, are very often determined by an injury.

There is yet another point to be noted in this case. The child has slight wrist-drop on the right side; he has, in other words, weakness of the muscles supplied by the musculo-spiral. Probably this also is the result of the injury. He is being

* ‘Leçons cliniques sur les Maladies du Système nerveux,’ tome iii.

treated by gradually increasing doses of arsenic, which is regarded by the profession as a specific in cases of chorea, and he will probably be well in a few weeks. It is unnecessary to devote further time to this disease, and we will pass to the consideration of two other conditions to which the term chorea has become attached, though, as we shall see, they differ considerably from that disease—hysterical chorea and post-hemiplegic chorea.*

2. *Rhythmical chorea* (Chorée. Rhythmée of Charcot, Hysterical Choreiform Spasms).—The clonic movements to which hysterical subjects are liable are numerous and varied. Almost every kind is met with, from a fine vibratile tremor to large convulsive movements. Tremors are less frequent than the larger movements, but I shall be able to show you at least one case on a future occasion. Various names, based upon the character of the movements, have been suggested, such as dancing chorea (saltatoric spasm), jumping chorea, hammering chorea, &c. But the term which I prefer for the indication of this group is rhythmical chorea, which I believe was first suggested by Charcot,† because the difference in the character of the gesticulations is not an essential one. The disease is said by Charcot and others to be very rare. It is certainly not very common, but I believe it to be much less

rare than is generally believed, and that cases are not infrequently confused with St. Vitus' dance.

I have notes of thirteen or fourteen cases, and before narrating three of these I will briefly indicate the characters which all these presented in common. First, the movements, when closely observed, are seen to be rhythmical. This feature, you will notice, is the one in which they differ most markedly from chorea. Secondly, they are generally "purposive," systematic, or expressive of some gesture. Thirdly, their distribution is generally more or less limited either to the head and neck, or to one or perhaps two of the limbs. Fourthly, they come on in paroxysms, though the movements in a smaller degree may be continuous.* Fifthly, these attacks or crises can generally be provoked by certain procedures, such as the pulling of a limb or pressing on the ovarian region. They may also occur spontaneously, or when the patient is startled. Sixthly, with the exception of one case, in which no note was made, the deep and superficial reflexes are markedly increased. Seventhly, all my cases have arisen in the female sex, and the patient has sometimes presented hysterical stigmata. Eighthly, the onset of the disease is usually sudden, and often dates from an emotional storm. I have been induced to mention those points because several of them are features to which attention has not been directed; and I believe the disease to be commoner than is generally supposed.

A case of this kind was sent to me some years ago by Dr. A. J. Masters in the person of a young lady twenty-four years of age. She was a country girl, but had been staying in town with the family of her lover for some weeks. The novel and amorous nature of her surroundings had evidently upset her nervous system. To such an extent was this the case that when the patellar tendons were tapped she almost jumped out of the chair ("strychninism"), and this procedure started jerking movements in all the limbs. However, what she was brought to me for was the rapid choreiform twitchings of the right upper extremity, more

* There is one other disease to which the term is applied, which is known as Huntingdon's chorea, or hereditary chorea. It is a somewhat rare condition. It affects adults from thirty to fifty years of age, especially women, and often runs in families. Its onset is gradual, and course prolonged through years in spite of remedies. The clonic spasms take the form of incoherent gesticulations involving all the muscles of the body, often in paroxysms which prevent the patient walking. They cease during sleep, and at first they cease during voluntary muscular action. The speech becomes stammering, the voice altered, and swallowing difficult. The ocular muscles are said to be exempt, but in one case I observed this was not so. In course of time the intellect is affected, and the patient becomes bedridden, tonic rigidity replacing the clonic spasms. It is distinguished from paralysis agitans by the involvement of the face and the range of the movements; from myoclonus multiplex by the movements being slower and more irregular.

In the only fatal case recorded basal pachymeningitis was revealed. If this be the lesion upon which such cases depend—and I have seen cases at the Paddington Infirmary which support this view—it would bring these cases into line with post-hemiplegic chorea. The partial damage of the cells of the cerebral cortex, which derive their nourishment from the meninges, would account for these as for other clonic spasms.

† 'Leçon's cliniques des Maladies du Système Nerveux,' iii.

* This occurrence in crises or paroxysms is very characteristic. A typical paroxysm commences with an aura of some sort, frequently an uneasy sensation in the epigastrium. This is immediately followed by clonic rhythmical spasms, which, becoming rapidly more and more severe, may finally end in tonic rigidity, the body or limb remaining for a few minutes in a strained position.

or less rhythmical, though of varying range. They had come on quite suddenly with a cramp-like pain in the hand after some unusual excitement two weeks before I saw her. They were almost continuous—"they cease occasionally," she said,—but were liable to severe exacerbations or attacks which came on when she was nervous or excited, or when the patellar tendon was struck, or the inguinal region pressed on. The movements involved the muscles of the scapula, upper arm, forearm, and hand. The movements ceased for a minute or two while she grasped anything firmly. The right leg was now and then affected with spasmodic jerks. There was no anæsthesia or other abnormality, and no neurotic manifestations in her history. Her family history revealed no neurosis, but her father had died of drink—another illustration of the power of alcohol to produce nervous disease in the offspring. She improved greatly under bromide, but she did not get rid of the movements until some weeks after her return to her quiet country home.

The patient who is coming in, a well-grown young woman of twenty-four years of age, is under the care of my colleague, Dr. Outterson Wood. Although of so masculine a build, she has always been very emotional, and subject occasionally to a feeling of a "ball" in her throat. Her family history is also neurotic. She was attacked quite suddenly a day or two after Christmas, 1896, with "eye-blinking," and a few days later these clonic spasms of the eyelids spread to the head, neck, and the upper extremity of the left side. When she came to the hospital in February these parts were the seat of continuous small, more or less, rhythmical movements. But they were also liable, when she was startled or disturbed in any way, to severe exacerbations, which were ushered in by curious inarticulate screams. I had the opportunity of seeing several of these attacks, and they were of great violence. The paroxysms can be provoked in the manner mentioned, but they also occur spontaneously. They commence quite suddenly, with a feeling of uneasiness in the epigastrium; then the patient gives a loud scream, or series of screams, her head is jerked violently to the right by a series of rhythmical clonic spasms, the left arm oscillates violently in the air, she rises suddenly from her chair, and the left foot is stamped violently upon the floor. The whole attack lasts five

or ten minutes, and then she sinks exhausted into a chair, though the movements continue in a minor degree. She is conscious, and can converse all the while; but she cannot control the movements, nor prevent bruising herself against the furniture.

There are three features of considerable interest about this case. First, although these attacks sometimes come on spontaneously, they can always be initiated by pressure upon either mamma, and conversely they can be completely stopped by firm steady pressure upon the left ovarian region, *i. e.* in the inguinal region of the affected side. This fact I pointed out to her and her sister, and they gladly employed it afterwards as a means to arrest the attacks, which were always momentarily preceded by the epigastric "aura" I have mentioned. Secondly, she underwent many various kinds of treatment at home, and she also spent several weeks in St. Bartholomew's Hospital, where she was visited by her father and other anxious relatives almost daily. But none of these means were of any avail, and it was not until she was sent right away to Scotland, and was completely removed from her home surroundings and her relatives, that she made any improvement. Then a speedy recovery ensued in the short period of a week or ten days, although she had been ill for over a year. Thirdly, you will observe as I tap her patellar tendons, not only is the tendon-reflex very exaggerated, but it brings on convulsive movements of both arms and of the neck muscles, similar in kind, though less severe than those just described. This, as I have pointed out on other occasions is an evidence of exaggerated reflex irritability, a condition of "strychninism" of the spinal cord.

It is upon this condition that the disorder, in my opinion, mainly depends, therefore the disease has not been eradicated; it is only dormant in her.

There are several other cases I should like to mention to you of this interesting disorder, but I think these are two fairly typical examples, though they do not always run this benign course; in one of Charcot's cases the attacks lasted for thirty years.

3. *Post-hemiplegic chorea* (post-paralytic clonic spasm, post-hemiplegic athetosis, &c.).—Paralysis due to lesions of the upper neuron is, in course of time, invariably succeeded, as you are aware, by rigidity or tonic spasm, and it is also followed, though not so constantly, by various kinds of

intermittent or clonic spasm. These spasms are of many different varieties—some observers make as many as ten or fifteen,—but they may be, I think, conveniently classed into three groups, which in order of frequency are (a) slow mobile spasms (athetosis), (b) smaller rhythmical tremors, and (c) irregular clonic spasms (which are called not inappropriately post-hemiplegic chorea when the movements follow hemiplegia). They may nearly always be recognised by the history of the antecedent paralysis, and by their being confined to the same distribution as that paralysis. These spasmodic movements are specially apt to come on after the paralysis of childhood and infancy, and especially when the paralysis has been slight or incomplete. When, therefore, the spasmodic condition has dated from childhood, the *history* of the antecedent paralysis may be wanting. This is especially so in the case of the children of unobservant mothers belonging to the lower orders. Here are two very good examples of the condition. The first of these cases is D. W—, aged twelve, a child who has had right hemiplegia since infancy. The history is somewhat wanting in this case; but you will see at the present time that her right hand and leg are weak and stiff, and that her right hand is the seat of slow athetotic movements, which are very characteristic. The other case is a woman named E. M—, aged forty-five, who is under the care of my colleague, Dr. Campbell. You will see that both the arm and leg of the left side are the seat of violent choreiform movements, which are almost convulsive in their character. The arm is affected more than the leg, and lately she says that the arm of the other side has become affected to some extent. She states that these movements have existed more or less as long as she can remember, but is unable to give us any further particulars. Nevertheless there is every reason to believe, from the study of similar cases, that she was in childhood or infancy the subject of left hemiplegia, which was gradually succeeded by these movements.

The study of these cases of post-paralytic spasm is very interesting, and throws considerable light upon the pathology of all clonic spasms due to organic disease. They prove, in point of fact, that clonic spasms are due to a partial or incomplete destruction of some part of the motor tract. These movements are relatively frequent after the

hemiplegia in children, in whom there exists, be it observed, a great recuperative power, so that the injured motor tract soon arrives at a condition of semi-recovery. When occurring in the adult they are relatively more frequent after embolic lesions, in which, be it observed, the damage is of wider extent, though of smaller degree, than in hæmorrhage, where the destruction is more complete, though usually of smaller area. They are also more common after lesions associated with hemianæsthesia, that is to say, lesions occupying the posterior part of the hinder limb of the internal capsule (the sensory crossway). Here, again, such lesions would be attended by less complete destruction of the internal capsule and motor tract than if they were situated further forward. These facts afford very strong evidence in favour of the proposition that clonic spasms due to organic disease are associated with an incomplete damage to the motor fibres, or, what amounts to the same thing, partial restoration. This statement is confirmed by the circumstance that in all cases of tremor where a lesion has been discovered, the lesion consists of partial or scattered destruction, as, for instance, in the senile degeneration of nerves in old age, which is so often attended by "senile tremor," and also in paralysis agitans.* Again, the scattered small lesions of disseminated sclerosis, or the scattered sclerosis and irritated (but not destroyed) cortex in general paralysis, are illustrations of the same fact. When speaking of small tremors I shall return to this matter again.

4. *Athetosis*.—The term athetosis is usually employed to designate a disease occurring for the most part in children and pregnant women. In this disease there are slow alternating flexions and extensions of the joints, attended also by a persistent or tonic rigidity of all the muscles of the body. The movements are less abrupt than most of the clonic spasms we have yet considered, and they are peculiar in being limited to the fingers and wrists, the toes and ankles. Occasionally the face and eyelids are affected. The patient is quite unable to hold anything in the hands, for there is, in addition to the clonic movements, a tonic rigidity of all the muscles—a painful stiffness. I

* Dr. Robert Maguire and myself have found a scattered partial degeneration in some of the motor nerve trunks in paralysis agitans.

am strongly of opinion that it arises from a toxic condition of the blood, for, amongst other reasons, it is always associated with marked gastric derangement. But we must postpone the discussion of this matter to a more appropriate occasion.

5. *Myoclonus multiplex* (or para-myoclonus multiplex), which appears to be allied to hysterical choreiform spasms, is an extremely rare and ill-understood condition. From the descriptions given of it, it appears to consist usually of sudden shock-like clonic spasms, of widely differing severity, bilateral, but affecting all the muscles of the limbs *excepting those of the hands and feet*, occurring mostly in adult males, running a chronic course, with a tendency to recover under appropriate electrical treatment. The most curious part of this strange disorder is the exemption of the wrists, ankles, hands, and feet. It is usually relieved by a fairly strong galvanic current, but is apt to relapse. This is the picture given of the condition in the books.

Dr. Bolton Thomson, of Luton, has very kindly sent up a case for us to see which he regards as an example of this curious disorder. It is that of a man, J. S—, æt. 63, who was formerly in the navy, but was invalided no less than thirty years ago, when this disease began with stiffness of his legs. During all that long time he has been affected with stiffness of the legs, and, in a less degree, of the arms, always worse in cold, damp weather. Moreover, by careful examination, you will observe that all the muscles between the knee and the ankle of both legs are affected by small, flickering, intermittent contractions. This symptom, the patient states, has also existed all this long while. This muscular “flickering” does not involve the whole of a muscle at once, but several bundles of fibres at a time, and is visible as a kind of rapid peristaltic wave beneath the skin, passing down the muscle. The same tremulous flickering can be observed in a less degree in the thighs, and he informs us that in cold weather the arms also are affected. These tiny tremors continue day and night, and only occasionally cease during sleep, according to his wife’s testimony. In addition to those rapid, intermittent contractions, the muscles of the legs, thighs, fore-arms, and upper arms are all slightly stiff. Myotatic irritability is also everywhere notably increased. The knee-jerks are

unduly brisk on both sides. Both the stiffness and the flickering become worse after standing or walking for some time, and he is never able to walk, at the most, for longer than an hour at a time. The muscles of the feet and hands, as you see, appear to be normal in every respect. He states the arm muscles have been getting weaker and thinner quite lately, but there is no notable wasting anywhere. His father died in an asylum, and one sister is in one of those institutions now; but his own mind is clear enough, and he enjoys remarkably good health for his age. There is nothing in his history which will throw any light on this strange condition. Galvanism does not seem to have relieved him in any notable degree.

What the precise nature of this case is, it is impossible to say. It does not appear to me to correspond with Friedreich’s original description of *Paramyoclonus multiplex*.* The vibration tremors—although involving the same muscles as that disease—are much too small; and they affect the individual muscular fibres rather than the muscles and groups of muscles. I have shown it to you in association with that disease because it has been so classed by others, but I think we must be content to leave it unnamed. It is one more illustration of the fact that nature—whether in health or disease it matters not—refuses to be pigeon-holed. I have never seen a case at all resembling this patient but once, and that, curiously enough, was quite recently. The patient was a gentleman about thirty years of age, who was shown to me by Professor Gairdner. All the muscles of his legs, more in the calves than the thighs, were affected, after walking any distance, with fibrillar tremors precisely resembling those you have seen to-day in the other case. They only came on after walking a mile or two, or riding a bicycle for five or six miles, and were accompanied by a tired, aching feeling. The trouble had come on quite gradually during the preceding three or four years, and as he had got worse lately he was afraid that some grave disorder would supervene. Bromide of ammonium gave him temporary relief.

In conclusion, gentlemen, you will have observed that there is a marked difference between the chorea of Sydenham and the other generalised

* Virchow’s Archiv, Bd. lxxxvi, p. 421.

choreiform movements we have been considering, in the age of the patients and the course which they pursue. The former runs a definite course, and is practically confined to childhood, excepting in the rare instances in which it is associated with pregnancy; but the latter are almost entirely confined to the adult. If, therefore, you meet with a case of choreiform movements in adult life, the patient not being pregnant, the diseases you would suspect are hysteria (rhythmical chorea), post-hemiplegic chorea, myoclonus multiplex, Huntingdon's chorea, and perhaps athetosis.

You will observe, also, that only the second named of these is definitely associated with an organic lesion. The others are what are called functional; or may it not be that they are of constitutional or toxic origin? At any rate we see by a close study of the clonic spasms of known organic origin that they are due to a *partial* or incomplete damage to the motor tract. We shall find in the course of our studies further proof of this. Having established this we will inquire what is the nature of this damage, and I shall hope to submit to you facts pointing strongly to the view that in a considerable number of cases it is of a toxic or nutritional kind.

Next time we shall turn our attention to a group of localised clonic spasms, such as habit spasm, spasmodic wryneck, and reflex muscular twitchings.

X Rays in the Army.—Dr. William M. Gray, the microscopist of the Medical Museum at Washington, was detailed by that institution for surgical work in the war, with special reference to the diagnosis of gun-shot wounds by the Röntgen rays. In giving the results of his observations Dr. Gray says, "We took out bullets by the pint on board the 'Relief,' and almost without exception they were located by the X rays. It is all done in a few moments: five seconds for a wound in the hand, thirty seconds for one in the foot, and not over ten or fifteen minutes for a wound through the pelvis. The patient is stretched out on a table, the X-ray bulb adjusted over the wound, the plate put under the limb or part where the wound is, and the thing is done. The plates are developed almost instantly. In many cases we save hours of vain searching; not infrequently we save the soldier's life."

Cincinnati Lancet Clinic, Oct. 1st, 1898.

CHAPTERS FROM THE TEACHING OF DR. G. V. POORE.

No. X.

GENTLEMEN,—You must remember that poisoning is a common delusion of the insane, who say they are being poisoned, and that their friends, often members of their family, are putting poison into their food or drink, and so forth; and sometimes it is a difficult matter to deal with these people. I once came home in the afternoon and found a box had been left, and that the person would call for it again. It was locked, and I did not at all approve of having a box of this kind left in my house by a stranger; therefore I called in a policeman and broke it open. It was filled with jars and saucers containing slices of mutton, vomit, fæces, and urine; and presently the lunatic to whom the box belonged turned up. It was a woman who thought she had been poisoned. I referred her to a chemist, and told her that if she wanted the things analysed the fees would probably be ten guineas for each analysis. The analyst never heard anything more of it, nor did I. Sometimes you can check silliness by stern realities. When you suspect poisoning I need hardly say you will watch the case with great care, and you will note the relations of the symptoms to the meals and medicine, and you will note the times of exacerbation and remission. And if death takes place you will notice the exact time of death. If you suspect poisoning you must take a note of all explanations offered to you and the circumstances, and you must be careful to reproduce the exact words used. You will also inquire into the nature of the food, into the cooking, the utensils, by whom the food was eaten; preserve all suspicious food, powders, bottles, &c., and preserve vomited matters. Sometimes a doctor may be put in a very delicate position, and if you believe suspicions which are ill founded you will easily get yourself into trouble. Now if you have those suspicions you will always do well to seek a consultant and tell him what your suspicions are. You must tell the friends that you must make inquiries as to the cooking and storing of the food, and whether there are any unwholesome condi-

tions. You would not mention poisoning, but you would say you were not quite easy whether the trouble was not caused by something in the food. If there is anybody in the house giving any poison, your action will excite their suspicions and do some good. I need not say to a body of sensible men that to breathe the idea that there are murderers in the house is as serious as anything well could be. The detection of poison in the vomit, or the detection of poison in the urine, of course would be a very important matter. I reminded you yesterday that certain diseases resemble irritant poisoning—cholera, acute dyspepsia, gastritis from whatever cause, perforation of the stomach, intussusception of the bowels. And then there are certain conditions which resemble narcotic poisons, such as apoplexy and epilepsy, and tetanus is very like strychnine poisoning.

Next with regard to convulsions. Convulsions do not occur without cause, and convulsions are very common as a result of stomach irritation, especially in children. It is said that the child has fits as easily as the adult has dreams, and I think it is fairly true. Indigestible food gives an adult uneasy dreams or nightmare, and the child not only has that but convulsions also. One of the worst attacks of convulsions that I ever saw was that of a child who was in the status epilepticus for hours, and it was due to the swallowing of a piece of slate pencil. Note that. It was nothing in the blood, nothing in the brain, it was purely the irritation of a foreign body in the stomach, insoluble and inabsorbable. It seems to me most important to remember such a possibility, because I think the tendency in the present day is for neuro-pathology to be too exclusively central. Now a person having died of poisoning, we have to consider how long the symptoms have lasted, and that brings me to speak of what is called the fatal period of a poison. After taking a lethal dose of a particular poison, how long may a person live? You will find the fatal period stated in books on medical jurisprudence, but it is so very uncertain that I do not think we can lay down any absolute rule. When we come to particulars I can give you extremes of recorded cases, but it is impossible to lay down any exact rule. Take such a poison as prussic acid, for instance. If it be as strong as the art of the chemist can make it,

prussic acid kills instantly it is said, just as if a man were shot by a bullet. But if we have prussic acid kept in a pharmacy or in a doctor's surgery in the country, it steadily deteriorates. Therefore in one case a man may die instantly, and in another case he may not die at all, or he may only die after many hours. Again, in this connection I would caution you, as I have in connection with other subjects, not to attempt to be precise where precision is impossible.

Now as to the post-mortem appearances in poisoning. Sometimes you will find a discussion as to whether the post-mortem appearance of the stomach is due, for instance, to arsenical poisoning or to gastritis. That is nonsense, because arsenic causes gastritis, and if you find gastro enteritis, what you have got to determine is, what was the cause of that gastro-enteritis; was it a poison or something else? I show you a preparation of a stomach; you see it is congested; and remember it is not the stomach of gastric irritation nor of poison, but of chronic heart disease. When such a poison as arsenic has been taken, if it has been taken in the solid form you find that wherever a speck of white arsenic is attached to the stomach, the mucous membrane round it is swollen and injected, but you cannot be too careful in coming to conclusions of this kind. I remember once making a post-mortem on a lunatic who had died in a private asylum. Associated with me at the post-mortem was a gentleman who was then and is still one of the best pathologists and morbid anatomists in the country. We found in the stomach little patches of inflammation, and the mucous membrane was gone in places. We came to the conclusion that the lunatic had been fed by the stomach-pump for weeks before, and that the pump had probably caused a little damage here and there.

Then remember that inflammation is known by local injection of the vessels, swelling of the mucous membrane, and the products of inflammation, either cloudy swelling of the mucous membrane or great secretion of mucus or secretion of pus. Again, remember to distinguish between post-mortem softening and inflammation, and perforation. When people die suddenly or are killed, that is drop dead in a state of good health, and when the stomach is secreting gastric juice at the moment of death, and if the gastric

juice lies in the stomach you get post-mortem digestion of the stomach, and perforation may be caused by that. I think if care is exercised it is not possible to mistake one condition for the other. Here are the most beautiful drawings by Sir Robert Carswell; these drawings will remain true for all time. Here is the stomach of a rabbit perforated by post-mortem digestion. There is no injection of the vessels, no swelling around the margin of an ulcer, but a gradual thinning away of mucous membrane to the spot where the gastric juice has acted best. I need hardly remind you that there are certain conditions which lead to perforation, such as chronic perforating ulcer of the stomach, or cancer; there is a perforation which is met with after burns; and there is the perforation of the intestine so common in enteric fever, and not uncommon in tuberculous disease of the intestines. Then in making a post-mortem on a case of poisoning, or where you suspect poisoning, certain precautions are necessary. First as to the contents of the stomach. When the stomach is opened, notice the odour instantly. Occasionally when you come to the dead body you may detect the odour of the poison which has been taken, such as prussic acid, carbolic acid, or perhaps phosphorus or opium. In opening the stomach you would do well to pour the contents on to a big photographer's dish, and look at them with your naked eye and with a lens, and if there is undigested food in the stomach you will be able to say what the food is, whether it is vegetable or not, and you will know what the patient has eaten. For instance, in one case a large amount of obvious onion was found, and in that particular case the man had been deliberately poisoned by a person who gave him roast duck for dinner, and had poisoned the stuffing and not the duck. The presence of hardened fæces in the intestines would show that no diarrhoea has taken place, it is said. But I think that is rather too strong a statement, because liquid fæces may find their way past scybala and be discharged. In cases of poisoning you must examine all organs with the greatest care, because the counsel for the defence will exert all his ingenuity to find out what you did not do. You must be careful to be able to say that there was no cause for disease elsewhere than in the stomach.

Sometimes you have to make a post-mortem on

an exhumed body. When that is the case there must be somebody present to identify the body. When exhumation is practised it is customary to remove some of the earth round the coffin, because it has been said in defence that the mineral poison found in the body was really present in the earth alongside. You must also be exceedingly careful if you have to preserve fluids or the contents of the stomach. There is a case on record, I believe, in which the contents of the stomach or the stomach itself, and some of the viscera, was carelessly wrapped up in a piece of wall-paper, there being nothing else at hand. This paper was printed with arsenical pigments. Then a case broke down because, not being provided with perfectly clean vessels or vessels of any kind, the doctors sent hurriedly to the village shop for a brown jar, and it came out in the evidence that the man who sold the bottle dealt largely in arsenic which was used for sheep-wash and for mixing with corn before it is sown. Of course counsel for the defence made the most of that circumstance. A further important point is, when you put the contents of a stomach into a vessel, be sure you do not put any preservative fluid with them. Probably a glass stopper is the best thing to close a jar with, and the next best thing is a bung covered on the inside and outside with new gutta-percha tissue. Then you must tie and seal and label the vessel. For tying down bottles such a thing as tinfoil, which is largely composed of lead, should not be used, as it may complicate the analysis. Again, the bottle should not be too big, the tighter the fit the better.

Another point is that poison may be found post mortem and the person may not have died of it. For instance, a case of splenic anæmia may have been taking Fowler's solution. Not only may poison be found in the body and the person not have died of it, but persons may be convicted of poisoning although no poison is found in the stomach. That was the case with William Palmer, who poisoned his victim with strychnine. That was forty years ago, when the mode of detecting strychnine was not properly understood. The victim had vomited a great deal, and the presence of strychnia in deceased's stomach was not clearly established; nevertheless the symptoms were perfectly clear, and the fact that the murderer had bought strychnine was clear, and the circumstances

brought home the crime to the murderer unflinchingly. Before making an analysis you ought always to have something to guide you. If you want to succeed you ought to know what to look for, and wherever it is possible you ought to have a statement of the patient's symptoms, whether they were narcotic or tetanic, or whether there was much vomiting and gastric irritation. There is only a limited amount of material to be dealt with, and it is useful to have a clue.

When cases of poisoning come before juries they like to know not only that a poison was present, but that it was present in a fatal amount—that a lethal dose was present. It stands to reason that a man may be poisoned by arsenic, but he may have vomited and got rid of much of it, so that there may not be a lethal dose in his stomach. That has happened again and again and again. Nevertheless when an analysis is made it is very desirable for practical purposes to establish the presence of an amount which is reasonably sufficient to kill. With regard to amount, it has been said that large amounts of poison may be taken to indicate suicide and not murder; the suicide, it has been said, is the person who takes large doses and uses double-shotted pistols, not a murderer. There is something in that, but these speculations you will find have always turned up at notable trials where there has been a large sum of money spent in the defence and counsel have racked their brains for every possible explanation. In the Madeline Smith trial eighty-eight grains of arsenic were found in the victim's stomach and other viscera, which is an enormous amount to find, and the idea was put forward that being a large amount it was of course suicide. Then chemistry is sometimes of importance in other ways. In a case of poisoning with sulphuric acid, for instance, it was alleged that the poison had been put into the coffee made for breakfast, and the coffee was made in a common metal coffee-pot. But when the coffee-pot was examined there was no evidence of sulphate of iron, so that it is hardly possible that sulphuric acid had been put into the coffee. Occasionally the analysis is of value from the negative point of view. People are suspicious that this, that, or the other poison has been given, and an analysis of the food and vomit has revealed nothing to justify their fears.

Occasionally it happens that physiological testing is more valuable than chemical testing. Undoubtedly it was the combined physiological and chemical testing which led to the conviction of Lampson for giving aconitine. First of all it was separated from the stomach, and then it was known that not only was it fatal to small animals but that it caused aconitism, tingling of the lips, &c., when some of it was put upon the tongue. Then it is said that the physiological test for strychnia, that is to say putting some of the extract on the back of a frog, is more delicate than the chemical test. So with atropine; putting a drop of the extract of the stomach into a cat's eye to make the pupil dilate is said to be more delicate than a chemical test. But in physiological tests of this kind you must be careful against arguing from animals to man, because there are some animals which seem immune to certain poisons. The most notable of these, perhaps, is the rabbit and atropine. Rabbits seem able to browse upon belladonna without harm.

Then there is another possibility. You must bear in mind that the flesh of poisoned animals may be poisonous to human beings. Sometimes the detection of a poison is made *via* the lower animals. For instance, there is on record a case where a man vomited into the chicken yard and arsenic was found in the chickens.

So much for generalities, except a few generalities as to treatment of cases of poisoning. That, of course, is very important to you as medical practitioners; perhaps more so than it is to you as medical jurists. But the principles of treatment for poisoning are mainly three. When you are called to a case of acute poisoning your first duty is to get rid of the poison. Sometimes you find the patient vomiting and purged, and occasionally it has happened that the doctor, being called in to see the patient in this condition, and not recognising the fact that the vomiting and purging might be due to an irritant poison such as arsenic, has tried to stop the vomiting and purging by the administration of another poison—opium. Therefore, when trying to get rid of the poison be careful not to throw an obstacle in the way of its elimination. Then to get rid of the poison you may give emetics, and in giving emetics I would say that it is advisable, if you have a choice—sometimes you have no choice.

—to give emetics which are not likely to complicate the analysis.

Tickling the back of the throat with a feather or with your finger, or the use of hot water, will not complicate the analysis; neither would mustard and water, I take it. Possibly ipecacuanha would not. But sulphate of zinc, which is a powerful emetic, may complicate the analysis. Possibly apomorphia, hypodermically injected, does not complicate the analysis, but that is one of those things which is not very often at hand. When we come to speak of particular poisons you will see that there are reasons against this or that line of action, and there are very often reasons why the stomach-pump must not be used. The stomach-pump does not complicate an analysis; but, as I pointed out just now, it might complicate a post-mortem. Remember that in passing a stomach-tube in a hurry you may injure the mucous membrane of the organ, and traumatic inflammation may come on, which you must keep in mind.

Having first of all tried to get rid of the poison, the next thing is to stop its action. You can stop the action of a poison by rendering it less soluble and absorbable than otherwise would be the case. Perhaps the best example of stopping the action of a poison which I can give you is by the administration of white of egg when a person has taken corrosive sublimate into the stomach. The result of that is that the albumen of the egg is coagulated by the corrosive sublimate, and you get the stomach full of albuminate of mercury. You must remember that you have then albuminate of mercury, insoluble in water, but perfectly soluble and digestible in the stomach. If you leave albuminate of mercury in the stomach you get the symptoms of mercurial poisoning inevitably. One day a man was admitted into my ward who had been poisoned by the application of corrosive sublimate *externally* to a raw eczematous surface, and he had acute dysenteric diarrhoea, and was very bad. The then house physician, who was a very able and highly-educated man, for whom I have great respect medically, had given this patient white of egg with a view of its acting as an antidote to the mercury. It could not possibly do anything of the kind, because the mercury was not in the man's stomach, where it could meet the white of egg; the poison was in his blood, and, being in his blood, it had

albumen enough there. I put that to you because it was a revelation to me as to what a sensible man could do. The white of egg acted as food, and did no harm in this case; but under such circumstances it was no antidote. Now if an acid has been taken, you give an alkali to correct it, and if an alkali has been taken you give an acid; but there are limitations to that line of action, as I shall have occasion to show you when I deal with the poisons *seriatim*. Most antidotes, then, for stopping the action of poisons are chemical antidotes. But there are also physiological antagonists, and I would say that here we are on very much more tender ground. One of the most often quoted instances of physiological antagonism is morphia and atropine, but I shall show that the physiological antagonism of these two is probably not complete. When you give a physiological antagonist I would warn you to be very careful indeed not to give a lethal dose of your antagonist, because you are not acting on very sure ground; and a counsel for the defence, if he should find that the doctor in attendance had given a lethal dose of a second poison to stop the action of the first, would have, I think, a very good defence, and a very disagreeable defence for the doctor.

Schweiger: Lithiasis in Childhood ('Jahrbuch für Kinderheilkunde,' Bd. xlv, H. 2, 3).—Eight cases were seen by the author; in five the stone was in the bladder, in three impacted in the urethra. The symptoms were similar to the adult—painful micturition, retention, incontinence, hæmaturia, cystitis, and albuminuria (in one). For the removal of the stones the supra-pubic operation was performed. Concerning three cases, in which the stone was situated in the urethra, two suffered from acute retention and convulsions. In one a positive diagnosis could not be made with the sound. A resistance was encountered in the deep urethra. Nothing more definite could be made out. After a protracted warm bath, two stones the size of peas were passed. In the second case the stones could be felt through the perinæum, and were also passed, through the assistance of a warm bath. In the third case the scrotum, perinæum, and penis were swollen and painful. A fluctuating point in the perinæum was incised. With the pus removed there was a stone the size of a pea.—*Archives of Pediatrics*, October, 1898.

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